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CHRONIC PERICARDIAL DISEASE

Report of Twenty-Eight Cases of Constrictive Pericarditis

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INFECTION of the pericardial cavity may be followed by complete or partial obliteration of the sac, by extension of the reaction to the mediastinum and other surrounding structures with resulting adhesions, or simply by a variable number of benign adhesions which unite the pericardium and epicardium. This latter condition is usually undiscovered during life since it rarely if ever results in untoward symptoms. Because of these facts, simple asymptomatic adhesions will be dismissed without further consideration. Even generalized union of the epicardium and pericardium if nonconstricting may produce no significant disturbance of cardiac function.

The condition in which there are adhesions that connect the heart and pericardium to the surrounding structures is known as mediastinopericarditis. It is believed that most cases follow rheumatic fever. Some instances of mediastinopericarditis are of interest from the viewpoint of surgical therapy, and they will be discussed subsequently.

More interesting because of its frequency and better response to operative treatment is the condition in which the heart is compressed by dense scar tissue, usually known as chronic constrictive pericarditis. The major portion of this paper will be devoted to a consideration of this condition. Before beginning this, for the sake of completeness, one should mention the fact that there is an occasional patient without pericardial adhesions or scars in whom symptoms referable to the heart result from displacement of this organ by a chronic inflammatory process of the lung and pleura. It may be necessary to perform a thoracoplasty in order to allow the heart and great vessels to return to the normal position.

Constrictive pericarditis and mediastinopericarditis will be considered in the order named.

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CHRONIC CONSTRICTIVE PERICARDITIS

The treatment by operation of patients with chronic constrictive pericarditis constitutes one of the most gratifying chapters in the history of surgery of the heart. The subject is not new but it is only in recent years that the frequency of both the disease and its response to operation have been appreciated. This is demonstrated by the fact that Churchill (23) in 1929 was able to find in the literature the reports of only 37 cases in which operation had been performed. Since that

time, pericardiectomy for chronic constrictive pericarditis has been carried out on a combined total of more than 60 patients in the Massachusetts General Hospital in Boston, in the Lakeside Hospital in Cleveland, and in the Vanderbilt Hospital in Nashville. The diagnosis has been made in each of these clinics on a good many other patients on whom operation was not performed for various reasons. The reports in the literature indicate an increasing number of patients in whom the diagnosis is made and the scar is removed. For example, quite recently, the results of 7 such operations were reported by Heuer and 9 were described by Harrington.

DEFINITION

Chronic constrictive pericarditis may be defined as a thickening and contraction of the pericardium or epicardium or both to such an extent that there is interference with the normal action of the heart. It is generally agreed that the ability of the heart to dilate to the usual extent and hence to receive the normal quantity of blood during diastole is markedly diminished. It is likely that the emptying of the heart is interfered with to a lesser degree. The pericardial sac may be completely obliterated or there may be areas in which the two layers are not adherent. The pericardium may or may not exhibit areas of calcification; usually it does not. Small collections of fluid may be trapped between the thickened epicardium and pericardium. Important disease of the heart itself is rare except for atrophy of the myocardium in advanced cases. These structural changes by interfering with the movements of the heart and blood lead to a recognizable clinical picture which will be described subsequently. A photograph of an autopsy specimen is shown in Figure 1 and a photomicrograph of a section of this is given in Figure 2.

REVIEW

According to White (57), the first clear cut description of pericarditis was given by Lower in 1669. The condition was recognized by Lancisi in 1728, by Morgagni in 1761, and by Laennec in 1819. Hope in 1839 expressed the belief that endocarditis coexists with pericarditis in the immense majority of cases and reported that he had never examined an autopsy specimen of complete adhesion of the pericardium without finding enlargement of the heart. He wrote,

"Pericarditis, both acute and chronic, and especially that originating in acute rheumatism, frequently terminates in adhesion of the pericardium. Lancisi, Vieussens, Meckel, Senac, Corvisart, and more strongly than all Morgagni, are of opinion that, with a complete and ultimate adhesion, the patient cannot live in a state of health. I know not how it is that Laennec and Bertrin and Bouillard have formed an opposite opinion. The former states that he had opened a great number of subjects so affected, who had never complained of any derangement in the circulation or respiration; whence he infers that adhesion often does not in any respect interfere with the exercise of functions. Bouillard, in his later work, adheres though with a slight qualification, to the same opinion."

Hope continues in stating that his own experience is entirely opposed to this doctrine.

"Nor has the additional experience of seven years, since the preceding sentence was written, afforded me reason to alter my opinion. I have never seen an individual with complete adhesion of the pericardium enjoying the most flourishing health."

Hope apparently realized that tuberculosis may be the cause of pericarditis.

"In scrofulous and phthisical individuals, tubercles are sometimes developed in the false membranes of pericarditis and according to Laennec, this may cause the acute to pass into the chronic state, as frequently happens in the case of pleuritic and peritoneal false membranes."

Chevers in 1842 disagreed with Hope's statements concerning the frequent association of adhesive pericarditis and cardiac enlargement. It was his impression that Hope's views were correct only in those instances in which there was disease of heart valves as well as adhesive pericarditis. When the valves were healthy, Chevers observed a general diminution in the size of the heart and a contraction of its cavities. He described 4 patients with the autopsy findings in each case. All clearly had constrictive pericarditis. None had an enlarged heart. In 2 of them, tubercles were described. He states:

"It will be observed that in the cases which I have cited there is no evidence of the pericardial adhesions being owing to rheumatic inflammation: on the con-

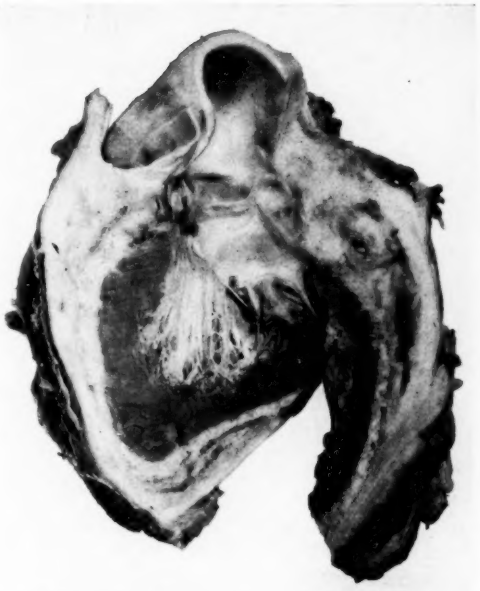

Fig. 1. This picture of an autopsy specimen shows the greatly thickened pericardium—epicardium overlying the left ventricle. It measured 3 centimeters in thickness at the base of the heart. The obliterated pericardial cavity is represented by the linear necrotic zone which is visible in some areas. The process was tuberculous in origin and activity was still present. Patient died less than 24 hours after entering the hospital.

trary, the adhesion of the lungs to the pericardium, in several, renders it probable that it depended upon severe disease of the idiopathic kind."

That Chevers had a good understanding of the disease is illustrated by the following:

"The principal cause of dangerous symptoms in cases of the above description appears to arise from the occurrence of gradual contraction in the layer of adhesive matter which has been deposited around the heart, compressing its muscular tissue, and embarrassing its systolic and diastolic movements, but more particularly the latter. Under these circumstances, the circulation seems, after a time, in great measure to adapt itself to the encumbered condition of the heart. The ventricles, having become diminished in capacity, make up for this loss by the rapidity of their contractions (hence the small and rapid pulse, noticed in three of the above cases): while the main arteries, if not already diseased, adapt themselves to the dimensions of the cavities from which they arise. . . . The heart had, doubtless, for a long time continued to become more and more compressed, weakened, and embarrassed, by the gradual contraction of the adventitious structure

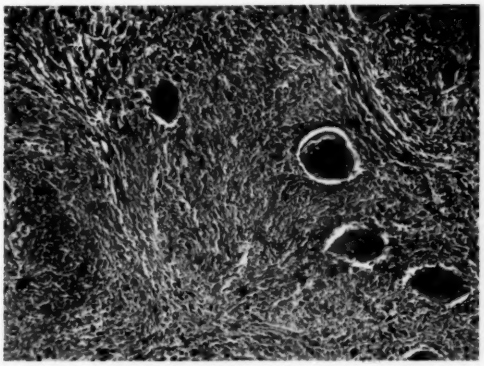

Fig. 2. Photomicrograph of scar shown in Figure 1. Many giant cells are present. ×100.

which surrounded it: distention of the great veins and abdominal viscera had necessarily followed; and the resulting anasarca, ascites, and effusion of serum into the pleura, in the second and third case, and also into the air cells of the lungs in the second, must have added still more to the obstruction with which the already almost powerless heart had to contend."

Concerning pericarditis associated with valvular disease, Chevers stated:

"It is evident, that if morbid contraction of any part of the valvular apparatus of the heart occurs coincidently with the first adhesion of the pericardial surfaces, the cavities behind the obstacle will have begun to suffer consequent dilation and thickening long before the adhesions have become so contracted as to exert much pressure upon their walls. And I am not prepared to maintain that a simply adherent state of the pericardium could materially check the occurrence of dilatation, should valvular contraction be set up at any subsequent period; but I must repeat, that it does not appear to me that we are at present in possession of the slightest definite proof that mere adhesion of its surface has any tendency to produce either dilatation or thickening of the heart."

Chevers found that there were recorded between 1835 and 1842, in the Museum Inspection Books, accounts of 47 cases in which adhesions of long standing were found uniting the pericardial surfaces. In 30 of these, the adhesions were general.

"The fact, that in nearly one half of the cases of partial adhesion the heart retained its ordinary dimensions, while in the remainder there was sufficient valvular disease to account for any enlargement that was present, would tend to prove that partial adhesion of the pericardial surfaces is not more liable to

Fig. 3. Rheumatic pericarditis, to be contrasted with the greater amount of exudation and fibrosis in tuberculous pericarditis in Figure 4. ×5.

Fig. 4. Tuberculous constrictive pericarditis with activity. ×5.

produce hypertrophy with dilatation than is the more complete form of the disease."

Wilks in 1871 agreed with Chevers in his conception of constrictive pericarditis. He remarked:

"I give six examples of cases where death was due apparently to adherent pericardium, and in looking through our post-mortem records I find that I might have added two or three more from every annular return. Therefore the condition cannot be regarded as being a rare one. A perusal of these cases would almost incline me to offer the proposition that in a well marked case of disease with cardiac symptoms in young persons without any valvular bruit pericardial adhesions may be fairly expected. In older persons, of course, we should look rather to degeneration of muscular tissue."

In 1873, Kussmaul called attention to a decrease in pulse amplitude in inspiration. Pick in 1896 gave a clear description of the differentiation of primary cirrhosis of the liver and chronic pericarditis. The disease is frequently designated by his name but this usage can hardly be considered correct in view of the previous excellent descriptions. The probable reason for the attachment of Pick's name to this condition is that it was at the time of his description that Weill and Delorme suggested resection of the pericardium for the relief of chronic constrictive pericarditis. Carl Beck in 1901, made a similar suggestion. This operation was first attempted by Henle in 1907 on a patient of Volhard's. The first successful result was reported by Rehn in 1920, the operation having been performed a number of years previously.

At the time that Delorme was pleading for the employment of surgery in the treatment of constrictive pericarditis, a great scientist, the founder of modern light therapy, Niels Ryberg Finsen, suffered from the disease. A recent consideration of his illness is given by Hugo Roesler. Finsen became ill at the age of 23 and was partially incapacitated during the remaining 21 years of his life. The findings on physical examination included ascites, pleural effusion, enlarged liver and spleen, normal sized heart, slight peripheral edema, cyanosis at times, and increased venous pulsations in the neck. He wished to avoid abdominal paracenteses and he reduced the rate of accumulation of ascites by low fluid intake, magnesia and ammonium chloride. He investigated extensively on himself the problem of water and salt metabolism and he placed himself on a low salt intake. His death occurred the year following the receipt of the Nobel prize. At the autopsy (performed at his request), extensive pericardial disease with calcification and an absence of valvular disease were found. The history of pulmonary hemorrhages suggests that the disease was tuberculous in origin.

The description of constrictive pericarditis by Volhard and Schmieden in 1923 is classic. It represents the greatest advance in the understanding of the mechanism of the disease since the description by Chevers in 1842. Volhard made a number of important points. He pointed out that simple adhesions produce no symptoms while the thickened adhesions cause symptoms because the heart loses its capacity

Fig. 5. Infra-red photograph of patient of Stewart and Heuer. a, Preoperative photograph; b, 3 weeks after pericardectomy; c, 4 months later; d, 10 months after pericardectomy; e, 17 months after pericardectomy. The progressive decrease in the prominence of the veins, the disappearance of the ascites and the change in shape of the chest are to be noted. (From Stewart, H. J., and Heuer, G. J. New York St. J. Med., 1939, 39: 2183.)

to dilate sufficiently to carry out its work. *Accretio cordis* or mediastinopericarditis, in which the primary disturbance is in systole, was differentiated from *concretio cordis* or constrictive pericarditis, and it was stated that there are intermediate types. Volhard stressed the disproportion between the maximal signs of peripheral congestion and the minimal signs of abnormality on examining the heart itself. The venous pressure was measured directly and the small heart in the presence of venous distention was commented upon. Volhard said that the neck veins collapse somewhat in both systole and diastole. He considered the increased pressure in neck veins as a most important point in differentiating the disease from cirrhosis. Seven patients with constrictive pericarditis were described by Volhard and good results were obtained in the 2 operated upon by Schmieden.

Youmans in 1926 reported 2 instances of calcification of the pericardium diagnosed during life and considered 94 collected cases in which the condition was found at autopsy. Coller incised in several directions the thickened pericardium of one of their patients. Definite improvement resulted despite the fact that no scar was removed.

Churchill (23) in 1929 reported the first successful resection of the pericardium for constrictive pericarditis in the United States and stimulated a still growing interest in the problem of the disease in this country. The second successful result was reported shortly afterward by Bigger (8).

Beck and Griswold (5) reported in 1930 the successful production of constrictive pericarditis in dogs by introducing Dakin's solution into the pericardial cavity. The picture is practically identical with that observed in patients. This method not only permits the obtaining of additional information concerning the mechanics of the disease but also allows one to secure added practice with the performance of a very delicate operative procedure which is carried out for the relief of the condition.

There has been careful analysis of a group of patients by White (57), admirably recorded in the St. Cyr lecture and there has been a series of studies on the circulatory dynamics of patients by Beck and Griswold (5), Burwell and Strayhorn (21), Burwell and Flickinger (19), Burwell and Blalock (18), Stewart, and others. An important review of the subject was presented by Schur in 1934.

Fig. 6. Photograph of autopsy specimen showing changes in inferior vena cava and hepatic veins. The viscera are seen from behind. The lungs have been removed and the aorta and inferior vena cava opened posteriorly. On the right the inferior vena cava can be seen passing from below upward to the "pars hepatica," where there is marked constriction, and into the right auricle of the heart. The two large, most inferior openings are the renal veins. The hepatic veins are of small size and there are thrombi in some of them. Two large hepatic veins opening above the constriction show narrowing almost to the point of occlusion. Note the greatly dilated veins on the dome of the diaphragm at the right.

PHYSIOLOGICAL CONSIDERATIONS

Many of the physiological mechanisms that are important in the manifestations and treatment of this condition were understood and described by Cohnheim 50 years ago as a result of his studies on the effects of fluid in the pericardial cavity. In considering the signs and symptoms of constrictive pericarditis, it is interesting that most of them are dependent upon two changes in the dynamics of the circulation. Studies that were carried out by Burwell, Strayhorn, Flickinger, Bowerman and others (18) have shown clearly that there are two simple mechanical disturbances behind the symptoms and signs which distinguish this condition. First, there is a diminution of the amount of blood pumped by the heart per minute and per beat and an inability to increase the output per beat. This diminution leads to weakness, fatigue, tachycardia, low pulse pressure, and a limited tolerance for exertion. The second mechanical disturbance is the increase in systemic venous pressure. The term "systemic" is used because the pressure in the pulmonary veins in patients cannot be measured and because various findings indicate that the congestion is more severe in the systemic than in the pulmonary area. Quite aside from any other change, an increase in venous pressure in itself can lead to venous distention, to engorgement of the liver, to the formation of peripheral edema, to nosebleeds, and to the transudation of fluid into the pleural and peritoneal cavities. The spinal fluid pressure rises with the venous. It has been found in experimental animals (12) that the lymphatic pressure also rises with the venous pressure. The alterations in the circulation were described by Heuer and Stewart as follows:

"Briefly, it was found in this group of patients that the arteriovenous oxygen difference was increased, the venous pressure elevated, and the circulation time prolonged; while the cardiac output per minute, the stroke volume and the cardiac index (cardiac output in liters per square meter of body surface per minute) were diminished."

Thus, the alterations in constrictive pericarditis include changes in the venous pressure which are similar to those in congestive heart failure and changes in the cardiac output and blood pressure which are somewhat similar to those observed in patients in shock. In the latter condition, however, the veins are usually

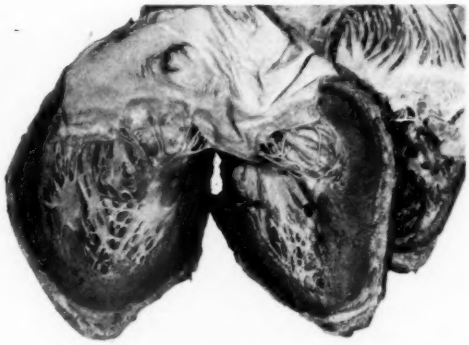

Fig. 7. Autopsy specimen, tuberculous pericarditis without fluid but with activity. This is the stage of the disease in which it has been necessary to operate upon most of our patients. Evidences of compression were not very marked in this patient.

collapsed, in sharp contrast to the distended veins seen in constrictive pericarditis.

Perhaps the alteration which is most responsible for the production of signs and symptoms is the marked increase in venous pressure. It has been observed by Burwell and Blalock (18) that marked improvement may follow a reduction in the venous pressure with little or no increase in the output of the heart. Usually, however, the output of the heart increases following operation as the venous pressure falls and it is impossible to place the blame accurately. The dyspnea that most patients with constrictive pericarditis experience on slight exertion is presumably due to the inability of the heart to increase its output adequately, and thus resembles the dyspnea suffered by normal persons after severe exercise. The edema, on the other hand, apparently is not due to the diminished cardiac output but rather in the main to the elevated venous pressure. This is suggested by the distribution of the edema, which is observed chiefly in the areas drained by the systemic veins (in which the pressure is much above the normal), while the volume flow of blood is necessarily reduced to the same degree in both peripheral and pulmonary circuits.

Schur is of the opinion that the extracardiac factors are important in constrictive pericarditis. He states if the only element were back pressure from the heart that the signs and symptoms would be identical with those encountered in any type of heart failure. There is marked interference with venous return in all cases of constrictive pericarditis and Schur believes this to be particularly true of the hepatoportal system. He says that the broadening of the hepatic vein orifices during inspiration does not take place in the presence of dense adhesions of the heart, diaphragm, and neighboring structures. He agrees with Schmieden and Fischer (49) that systole of the heart is also interfered with rather seriously. The heart is fixed at the base and at the apex, and it cannot shorten sufficiently to perform its work effectively. It appears, as has been stated, that the disabilities of constrictive pericarditis arise chiefly from the fact that the heart cannot dilate to a normal extent during diastole. One is also impressed by the disturbance of contraction and emptying which must be associated with an almost unyielding scar closely attached to the heart, and it seems likely that the heart could not put out a normal volume of blood per beat even if it could receive it.

Fig. 8. Autopsy specimen, tuberculous pericarditis, small amount of fluid, shaggy exudate. Little can be accomplished by operating in this stage of the disease.

ETIOLOGY

It is our contention, on the basis of evidence to be presented, that most instances of constrictive pericarditis result from infection with tuberculosis. It is impossible to be certain as to the etiology of many of the reported cases of constrictive pericarditis since a clear differentiation is not made in the majority of the articles between mediastinopericarditis, constrictive pericarditis, and simple nonconstricting adhesions between the pericardium and epicardium. In addition, if the scar is healed, microscopic study will not usually reveal the etiology of the disease.

Wells found adherent pericarditis of various degrees in 57 of 1,048 autopsies. Of these 57

cases, 8 were due to rheumatic infection, 6 were frankly tuberculous, and 43 were of undetermined cause. Regarding the last, he stated:

"By the above process of exclusion it has been attempted to show that many cases of adherent pericardium are probably of tuberculous origin, although not showing any anatomical characteristics of tuberculosis. However, it is to be understood that this is not the only cause."

It was his impression that the pericarditis in many instances was secondary to tuberculosis of the mediastinal glands. Concerning the differentiation of the rheumatic and tuberculous cases, he stated:

"In the rheumatic there is a total absence of tuberculous lesions in the mediastinal glands and lungs; the pleuritis existing is generally localized at the pleuropericardial surfaces, seeming to be secondary to the pericarditis; endocardial changes are usually present, in all but one of those recorded; death is usually due to cardiac failure in the rheumatic, while this is the exception in the healed tuberculous pericarditis; the age of the subjects is, on the whole, younger in the rheumatic."

Trout in 1931 collected all cases of adhesive pericarditis in which operation had been performed. The Delorme operation had been used in 43 of these and the Brauer procedure in 54. Concerning the etiology, he stated:

"It is usually regarded that the unknown cause of rheumatism, or, in the nomenclature of today, 'arthritis from focal infection,' is the chief etiologic factor in the production of these adhesions. However, in a brief review of some of the literature, I noted that various types of streptococci, pneumococci, and staphylococci, and of tuberculosis, gonorrhea, and influenza bacilli, etc., have been reported as being intimately connected with the causation of adherent pericarditis."

Smith and Willius of the Mayo Clinic found (at necropsy) 144 cases of chronic adherent pericarditis among 373 cases of pericardial disease. Only 57 of the 144 patients had presented complaints that made the heart the major issue in the clinical picture. The etiology was determinable in 73 of the 144 cases. Rheumatic fever was the etiological factor in 21.5 per cent, pulmonary and pleural disease in 17.4 per cent, cardiac infarction in 6.2 per cent, syphilis 2.8 per cent, neoplasms in 2.8 per cent, and tuberculosis in 2.1 per cent.

Schur in 1934 reported 17 instances of adhesive pericarditis, some of which had mediastinopericarditis and others constrictive pericarditis. Tuberculosis was suspected of being the etiological agent in 5 of these, in none of which operation was performed. Seven were believed to be rheumatic in origin. The etiological agent was not established in the first 6 patients upon whom pericardectomy was performed by Beck (4).

Rothstein in 1934 collected all instances of adhesive pericarditis in children under 15 in which the condition had been treated by operation. The total number of cases was 34. The etiological agent was thought to be acute rheumatic fever in 16, tuberculosis in 9, and pyogenic organisms in 2. The Brauer procedure was carried out in 23 of these, which would lead one to believe that the disease was mediastinopericarditis. In the 11 remaining, pericardectomy was performed. Four of these were supposedly tuberculous in origin. Borchardt states that rheumatism is the most frequent cause of chronic adhesive pericarditis and that tuberculosis plays a relatively smaller rôle. Tuberculosis as the cause of the constrictive pericarditis was proved in only 1 of the 7 patients reported by Heuer and Stewart.

White (57) had the following to state concerning the etiology of the 15 cases of constrictive pericarditis which he and Churchill reported.

"The etiology of the chronic pericardial disease can be assigned as follows: tuberculous in two (questionable in two others), pneumonia with polyserositis (including both pleuritis and pericarditis) in two, sepsis in one, rheumatism in none, uncertain or unknown in ten (in five of which, however, there was a definite history of acute pericarditis)."

White's report is similar to our experience in that none of the patients had a rheumatic infection. He stated:

"It is of further interest that a series of 1,000 children with the rheumatic infection studied in the House of the Good Samaritan in Boston, and followed over a period of ten years has shown, in not a single instance any evidence of chronic constrictive pericarditis, even though the heart was often seriously involved in other respects and even though acute pericarditis had been noted in many cases during their acute rheumatic infection."

Of the 28 patients with constrictive pericarditis that have been observed in the Vanderbilt University Hospital in the past 11 years,

the etiological agent in 21 was established by aspiration, operation, or autopsy. The etiological agent was the tubercle bacillus in 18 and the Staphylococcus aureus in 3 cases. In several others, there was suggestive evidence that the tubercle bacillus was the cause of the disease. Such a preponderance of tuberculosis as a cause of constrictive pericarditis is not confirmed by most of the reports in the literature. However, these patients had pericardial fibrosis which was constricting, while many of the statements in the literature are concerned with adhesions of all types.

The most striking generalization that can be made is that a constricting scar is a frequent result of tuberculous pericarditis and a rare result, if it ever occurs, of rheumatic pericarditis. In the latter infection, there is a sticky exudate of a fibrinous character which usually in a relatively short period causes the two surfaces of the pericardium to become adherent. The acute injury is present but there is not the prolonged insult such as occurs in a tuberculous infection. The differences in the pictures of the two are shown in Figures 3 and 4. In 3 of the cases here reported constriction occurred as a sequel to infection with pyogenic organisms. It appears that this is particularly apt to occur when operation for purulent pericarditis is delayed.

DIAGNOSIS

The diagnosis will be discussed in part by enumerating the findings in a group of patients in the Vanderbilt Hospital. These patients have been studied carefully by Doctors C. S. Burwell, T. R. Harrison, H. J. Morgan, David Strayhorn, and others. A total of 28 patients with undoubted constrictive pericarditis have been observed in the past 11 years. The diagnosis was confirmed at the time of operation in 20 of these, in 3 there was only the clinical diagnosis, and in the 5 remaining, the premortem impression was confirmed at autopsy.

The ages of the patients ranged from 14 to 77 years. The number of young patients in this series is less than that usually encountered. Only 4 of the 28 patients were females. Twenty were white and 8 were colored.

The patients have presented themselves for examination after illnesses varying from a few days to a number of years. Most of them had been ill for a number of months rather than years. In a certain number of cases the illness has begun with a respiratory infection, but this was not always so. Most of the patients came complaining of weakness, fatigability, swelling of the abdomen, edema of the feet, and discomfort in the upper abdomen. Dyspnea on exertion was frequent; dyspnea at rest was usually a late development if it occurred at all. On examination the impressive findings were those of systemic congestion; that is, distended veins, high venous pressure, engorged liver, ascites, and peripheral edema. Pleural effusion was frequent; pulmonary edema was rare. These signs of congestion often remained essentially unchanged for weeks or even months, unlike those in most cases of heart failure. This group of signs, which point so strongly to failure of the right side of the heart, were combined with a normal sized or only slightly enlarged cardiac dullness, a fixed heart, distant heart sounds, and an absence of visible or palpable apex beat. The heart rate was rapid and the rhythm regular. Heart murmurs were absent. The blood pressure and pulse pressure were usually low. The pulse was small and diminished or disappeared during inspiration in all patients except one. Under the fluoroscope, the pulsations of the heart were found to be greatly diminished or absent. Examination of the heart by the x-ray showed areas of calcification in some patients. In this connec-

TABLE I.—SYMPTOMS AND SIGNS IN 28 CASES OF UNDOUBTED CONSTRICTIVE PERICARDITIS

	Per cent
Distended veins	100
Increased venous pressure (180 to 390 mm. H_2)	100
Enlarged liver	100
Edema (peripheral)	96
Tachycardia	100
Diminished pulsation (fluoroscope)	100
Paradoxical pulse	93
Low pulse pressure	93
Faint heart sounds	96
Ascites	89
Pleural effusion	86
Greatly enlarged heart	0
Pulmonary edema (gross)	0
Hypertension	0
Valvular disease	0
Systolic retractions	0
Auricular fibrillation	4
Paroxysmal dyspnea	4

TABLE II.—CONSTRICTIVE PERICARDITIS

A. Patients Cured or Greatly Improved

Patient — Age—Sex	Duration of disability	Ascites	Heart rate — Temperature	Blood pressure mms. hg.	Paradoxical pulse	Liver size	Absence pulsation x-ray	Venous distention and pressure mms. H_2O	Pleural effusion	Etiology	Pericardectomy —Delorme	Result Remarks
1—E. Mc. 25—M.	5 mos.	5 mos.	100 98.8	110/80	?	++	+	+ ?	R	Healed scar	Yes	Cured. (Dr. Bigger) 12 years since operation
2—H. C. 17—M.	4 mos.	3 mos.	120 101	106/88	++	++	+	+ 355	R L	Staphylococcus aureus	Yes	Cured. 8 years since operation
3—E. E. 21—M.	6 yrs.	6 yrs.	120 100	90/60	+	++	+	+ 195	+	Healed scar	Yes	Cured. 7 years since operation
4—D. D. 28—M.	2–3 mos.	1 mo.	110 102	98/84	+++	++	+	+ 200	L R	Staphylococcus aureus	Yes	Cured. 7 years since operation
5—J. O. 18—M.	2+ mos.	+	110 100	100/80	++	++	+	+ 300	L	Tuberculosis	Yes Two	Markedly improved. 6 years since last operation
6—R.H.W. 61—M.	5 mos.	3 wks.	115 100	115/90	+	++	+	+ 215	L	Tuberculosis	Yes	Well. 5 years since operation
7—R. L. R. 30—M.	7 mos.	3 mos.	115 100	100/80	++	++	+	+ 220	R L	Tuberculosis	Yes	Cured of pericarditis, 4 years since operation. Bone graft for tuberculous spine. Cured
8—W.G.A. 57—M.	7 mos.	6 mos.	105 100	124/90	+	++	+	+ 180	R L	Tuberculosis	Yes	Markedly improved. Two years since operation
9—J. W. 17—M.	5 mos.	2 mos.	110 99	90/60	++	++	+	+ 300	L	Tuberculosis	Yes	Well, no symptoms, 2 years since operation
10—R. G. 58—F.	7 mos.	6 mos.	110 99.6	100/82	++	++	+	+ 220	R L	Tuberculosis	Yes	Markedly improved. Two years since operation
11—E. S. J. 25—M.	11 mos.	5 mos.	100 99.4	110/90	++	++	+	+ 290	R L	Tuberculosis	Yes	Markedly improved. 16 months since operation
12—W.B.D. 24—M.	3 yrs.	1 yr.	95 98.6	102/85	++	+	+	+ 350	R	Healed scar	Yes	Markedly improved. 20 months since operation

B. Patients Who Have Died Since Operation

13—C. W. 36—M.	7 mos.	2 mos.	110 99	118/104	++	++	+	+ 210	R L	Tuberculosis	Yes	Died 12 hours following operation from hemorrhage (Dr. Bigger)
14—J. W. 51—M.	4 mos.	7 wks.	110 102	105/90	++	++	+	+ 215	L	Tuberculosis	Yes	Improved for 6 months. Died miliary tuberculosis
15—L. C. 34—F.	3 yrs.	2+ yrs.	95 99	98/74	+	++	+	+ 250	L	Tuberculosis ?	Yes	Died 4 days following operation of pneumonia
16—B. C. 46—M.	6 mos.	+	90 101	90/80	+	++	+	+ 210	R	Tuberculosis	Yes	Died shortly after operation. Acute dilatation heart
17—E. C. 45—M.	4 mos.	3 mos.	100 101	108/84	+	++	+	+ 195	L	Tuberculosis	Yes	Died 2 months after operation of miliary tuberculosis
18—A. H.K. 40—M.	7 mos.	6 mos.	120 101	105/85	++	++	+	+ 278	L	Tuberculosis	Yes	Improved for 2 years. Died following reactivation of tuberculous process
19—E. B. G. 52—M.	8 mos.	4 mos.	110 100	120/110	++	++	+	+ 250	R L	Tuberculosis	Yes	Died 3 days following operation. Myocardial necrosis. Rupture of right ventricle
20—J. B. S. 14—M.	6 mos.	3 mos.	120 100	110/95	++	++	+	+ 265	0	Staphylococcus aureus	Yes	Osteomyelitis multiple, septicemia, acute and chronic pericarditis. Died 14 days after pericardectomy of bronchopneumonia

C. Clinical Diagnosis. No Operation

21—T. Y. 40—M.	13 yrs.	0	85 99	120/80	0	+	+	+ 210–280	0	Tuberculosis ?	No	Operation not advised. Disability only partial
22—M. R. 27—F.	6 yrs.	0	100 100	104/86	+	+	+	+ 210	0	Tuberculosis ?	No	Disability only partial
23—W. B. 45—M.	2 yrs.	3 wks.	120 100	115/95	+	+	+	+ 205	0	Tuberculosis	No	Bilateral pulmonary and renal tuberculosis. Died 2 weeks after leaving hospital

TABLE II.—CONSTRICTIVE PERICARDITIS—Continued

D. Clinical and Autopsy Diagnosis
Pericardectomy Not Performed

Patient — Age—Sex	Duration of disability	Ascites	Heart rate — Temperature	Blood pressure mms. hg.	Paradoxical pulse	Liver size	Absence pulsation x-ray	Venous distention and pressure mms. H_2O	Pleural effusion	Etiology	Pericardectomy —Delorme	Result Remarks
24—W. K. 40—M.	2 yrs.	+	80 99	98/84	+	+	Not done ?	+ 257	R L	Tuberculosis	No	Died one day after admission
25—J. B. 51—M.	2 mos.	+	120 103	120/100	+	+	+	+ 305	R L	Tuberculosis	No	In hospital 5 weeks. Was believed to have effusion but had scar
26—E. N. 50—F.	4 mos.	2 mos.	150 103	104/88	+	+	+	+ ?	R L	Tuberculosis	No	Died 13 hours after admission
27—S. N. 70—M.	5 mos.	+	110 102	94/60	+	+	+	+ 230	R L	Tuberculosis	No	Pericardium drained 5 weeks before death (Dr. Bigger). Pericardium 1 cm. thick at autopsy
28—A. D. 77—M.	2 yrs.	+	100 100	160/90	0	+	?	+ ?	L	Tuberculosis	No	Generalized arteriosclerosis. Slight cardiac hypertrophy and dilatation. Signs of constriction not marked

tion, it should be emphasized that calcification in the heart area may be due to factors other than disease of the pericardium. Further, calcification of the pericardium does not necessarily produce significant compression of the heart.

The electrocardiogram often showed small complexes and flat T waves. Heuer and Stewart described the following electrocardiographic changes in their patients:

"The electrocardiograms are of low voltage of QRS and T waves, and the latter may be 'cove' in form in leads I and II. The electrical axis may not shift or may shift only slightly, but too much emphasis is not to be placed upon this finding. There may be slight left or slight right axis deviation."

White (58) made the following statements regarding a comparison of the electrocardiogram in pericarditis and in coronary disease.

"The S-T interval and T wave changes resemble to a certain extent those found in coronary disease, especially those in acute occlusion, namely a low origin of the S-T interval with flattening or inversion of the Ts; there are, however, two distinct differences: (1) the T wave changes are usually consistently found downwardly directed in all three classical leads in contrast to the opposite direction of T_1 and T_3 after coronary thrombosis, and (2) the Q waves are not exaggerated in pericarditis as they are in either lead 1 or lead 3 after acute coronary occlusion. In the chest lead (Lead 4) the T waves in acute pericarditis tend to be upright instead of normally inverted."

The combination of a severe degree of peripheral congestion with a small quiet heart is the essential point in the recognition of constrictive pericarditis. Edema is out of proportion to the dyspnea. The peripheral signs suggest heart failure, but the heart itself supplies no basis for such failure. In a certain number of patients ascites appears before any other accumulations of fluid and leads to a diagnosis of cirrhosis or of tuberculous peritonitis. In all our cases, however, there has been evidence of obstruction to both the superior and inferior caval systems. Although the ascites is impressive, the neck veins are engorged and the venous pressure (after removal of the ascitic fluid) is usually as high in the arms as in the legs. The venous pressure remains persistently elevated and it is not altered greatly by the removal of fluid from the pleural or peritoneal cavities. A photograph of a patient of Stewart, Heuer, et al., demonstrating the venous distention, is shown in Figure 5. An analysis of the 28 cases shows that certain signs and symptoms are conspicuous by their presence and that certain others are conspicuous by their absence. These are shown in Table I. This table emphasizes the frequency with which the signs of peripheral congestion are found and the infrequency with which the signs of endocardial disease, hypertrophy, and pulmonary congestion are observed. It emphasizes the usefulness of the fluoroscopic examination and the determination of the venous pressure in the making of a diagnosis of constrictive pericarditis. Additional points of interest concerning these patients are given in Table II.

DIFFERENTIAL DIAGNOSIS

In most cases, constrictive pericarditis can be readily recognized and the diagnosis can be made with confidence. Mistakes, however, can be made, and the following conditions, among others, should be taken into consideration in the differential diagnosis of constrictive pericarditis:

1. Other types of pericardial disease,
   (a) fluid in the pericardium,
   (b) mediastinopericarditis,
   (c) polyserositis.
2. Diseases of the heart,
   (a) tricuspid valve disease,
   (b) diseases accompanied by failure of the right ventricle: mitral stenosis,
   cor pulmonale,
   myocardial disease (especially that due to coronary insufficiency).
3. Extracardiac disease,
   (a) cirrhosis of the liver,
   (b) mediastinal tumors,
   (c) nutritional edema,
   (d) multiple thrombosis of veins
   (e) conditions associated with marked increase in intrathoracic pressure.

1. Other types of pericardial disease. (a) Fluid in the pericardium may bring about cardiac tamponade with symptoms and signs (depending on rapidity of accumulation of fluid) similar to those of obstruction from pericardial constriction. If this is so, the fluid usually leads to changes in the size and shape of the heart-pericardium outline which are quite different from those seen in constrictive disease. When, during the course of subacute pericarditis, the heart-pericardium shadow becomes smaller and the signs of tamponade persist, the suggestion of the formation of a constricting scar is strong. Noth and Barnes observed differences in the electrocardiographic curve in acute and chronic pericarditis. In the acute form, there is often elevation of the RS-T segment and there may be exaggerated T-waves in the standard leads. In constrictive pericarditis, the T-waves are usually flat or inverted and the QRS complexes small.

b. Mediastinopericarditis is discussed elsewhere in this paper and will be mentioned only briefly in this discussion of the diagnosis of constrictive pericarditis. It is important to make a correct diagnosis between these two types of pericardial disease, since their treatment is quite different, and since the treatment of the constrictive form may be highly successful. It is suggested, especially in older literature, that these two forms of fibrous pericarditis are often associated, but this is not the experience of most observers in America. Mediastinopericarditis is usually a sequel of rheumatic pericarditis and is almost always associated with valvular disease (often multiple) and with marked cardiac enlargement. Both valve disease and cardiac enlargement are rare in patients with constrictive pericarditis. Finally, since in mediastinopericarditis, the heart is firmly bound by fibrous tissue to neighboring organs, there are signs which indicate the tugging of the heart on these adjacent structures. These signs, such as systolic retraction of the ribs and diaphragm, are not observed in characteristic examples of constrictive pericarditis.

c. Polyserositis, or the simultaneous inflammation of several serous cavities, is usually due to tuberculosis. It begins as an acute process with varying quantities of fluid and may progress to a chronic fibrous state. If the latter stage is reached, it may be difficult or impossible to differentiate it from chronic constrictive pericarditis due to other causes. In fact, we know that true constriction and polyserositis may occur at the same time. White (57) states: "Perihepatitis, perisplenitis, and even frosting of the intestine itself may occur quite independently of pericarditis or pleuritis; they may be found associated or as isolated lesions." The points of most significance in assessing the relative importance of inflammation and obstruction are the type of fluid (evidence of inflammatory origin) and the venous pressure.

2. Diseases of the heart. (a) Tricuspid valve disease brings about circulatory changes similar to those caused by constrictive pericarditis and therefore may be associated with similar signs. Both lesions offer an obstacle to the entry of blood into the heart, in one case an obstructive valve lesion, in the other interference with ventricular dilatation. Tricuspid disease is usually accompanied by other valve disease, by enlargement of the right auricle, and by veins which exhibit pulsation with ventricular systole.

b. Conditions accompanied by preponderant failure of the right ventricle include mitral stenosis, cor pulmonale, and occasionally myocardial disease. Right ventricular failure produces peripheral congestion similar to that of constrictive disease except that the congestion of muscular failure is more apt to fluctuate in severity than is that of mechanical obstruction. In addition, such cases of failure rarely exhibit pulsus paradoxus, and they usually show cardiac enlargement and other evidences of heart disease. A patient with cor pulmonale due to pulmonary vascular disease may show cyanosis, local signs of pulmonary disease, and a large right ventricle.

Myocardial disease may produce special difficulties, as exemplified by the following case:

H. M., white male, aged 44 years, entered the Vanderbilt Hospital on May 15, 1935, complaining of "abscess in chest." He was stabbed in the posterior part of his right chest 2 months previously with a long large knife. There was profuse bleeding from a sucking wound. The opening was closed and he remained in bed for a week. However, daily temperature elevations increased and he became dyspneic 3 weeks following the injury. Shortly following this, the old wound opened spontaneously and pus drained for a week. The shortness of breath was less marked following the drainage. In a few days, the wound again healed and subsequently opened spontaneously. The patient had lost 30 pounds during his illness. The past history was negative.

On examination, it was found that the patient had a chronic empyema on the right. In the right scapular line, there was a recent wound extending from the eighth to tenth interspaces with a small sinus near its center. There was a small amount of drainage from it. X-ray examination revealed hydropneumothorax on the right with marked fibrosis on the left. The empyema cavity was given adequate drainage by rib resection on May 17. He insisted upon leaving the hospital several weeks later, at which time the cavity held 100 cubic centimeters.

The patient re-entered the hospital on September 27, 14 weeks following his discharge. One week after having left the hospital, his physician injected some paste into the cavity. The opening closed shortly thereafter. The patient became short of breath 3 weeks prior to his return to the hospital and fullness of the abdomen and edema were noted. On examination, the patient was found at this time to be short of breath and cyanotic. The superficial veins were quite prominent, the pressure being 238 millimeters of water by a method rarely registering over 120 millimeters in a normal person. There was edema of the lower extremities and the sacral region. The pulse volume was small and distinctly paradoxical in character. The arterial blood pressure was 98/80. The heart was somewhat enlarged and showed very little pulsation under the fluoroscope. The sounds were distant and no murmurs were heard. The liver edge extended well below the costal margin. The impression of Dr. Hugh Morgan was as follows: "Pleuromediastinopericarditis. It seems probable that the obstruction to inflow of blood to heart is about the cavae and that we are not dealing with a generalized constrictive pericarditis."

An exploration of the pericardium was decided upon and this was carried out 4 days following his admission. The pericardium looked essentially normal and no abnormality was encountered except for the escape of a small amount of blood tinged fluid upon opening the cavity. There were no adhesions between the heart and pericardium. Incision was closed without drainage. Wound healed and patient's condition remained the same for 10 days when he became suddenly worse and died 2 days later.

An autopsy was obtained, and the following observations were made. The left pleural cavity contained 600 cubic centimeters of fluid and the peritoneal cavity 2,000 cubic centimeters. A pasty mass was found in the right pleural cavity. The pericardium was essentially normal. The superior and inferior vena cavae appeared normal. The heart weighed 550 grams, and the musculature was pale in color and flabby in consistency. There was a large mural thrombus on the posterior wall of the right auricle and also one about 3 centimeters in diameter on the posterior lateral surface of the left ventricle. On cut section the musculature at the apex of the right ventricle showed several small depressed areas about one-fourth centimeter in diameter which were of a dull red color. They were also present in the left ventricular wall. Microscopic study showed thickening of the endocardium and many phases of degeneration and necrosis of the myocardial fibers. There were large areas of fresh hemorrhage scattered throughout the myocardium and in these areas, necrosis of the muscle cells was pronounced. The muscle fibers were everywhere hypertrophied and the nuclei were large and vesicular. In most areas, however, they were fragmented and stained irregularly. The liver presented a uniform picture of acute necrosis of all the parenchymal cells surrounding the central veins. The impression from autopsy was that the patient died of cardiac dilatation and hypertrophy, myocarditis and endocarditis. This case is cited to show that some of the signs seen with constrictive pericarditis may be present in other conditions and to emphasize that it is not any single sign but a total picture that is distinctive.

A combination of right ventricular failure with a small amount of pericardial fluid led in another case to an incorrect diagnosis of constrictive pericarditis.

E. A., a white female, aged 35 years, entered the hospital on December 8, 1934, complaining of "swelling of abdomen."

Nine months previously, the patient noticed that her abdomen was increasing in size, and edema of the feet and face appeared. This was followed by shortness of breath. She became extremely weak and complained of palpitation and dizziness. She was treated with diuretic drugs and digitalis and some improvement followed, but the dyspnea and edema never disappeared entirely. There had been frequent attacks of epistaxis during the 2 months preceding her admission, and nausea had been present for a week. There had been a gain in weight of 13 pounds during the illness.

The face was swollen and the superficial veins of the entire body were unusually prominent. The retinal veins were distended. The pressure in veins of the arms and legs was 172 millimeters water. There was evidence of a slight increase in the size of the heart-pericardium by both physical examination and x-ray. The pulsations of the heart were indistinct under the fluoroscope. The blood pressure was 118/104 millimeters mercury and the pulse was believed not to be paradoxical. There was a good deal of fluid in the left pleural cavity. The liver extended seven fingers-breadth below the right costal margin in the midclavicular line and there was evidence of free fluid in the peritoneal cavity. The temperature was 101 degrees F. and the pulse 100. The electrocardiographic tracing showed (1) notched QRS in all leads, and (2) no shift in axis. The cerebrospinal fluid pressure was 210 millimeters water.

She developed a generalized macular rash which was associated with a cough and an increase in temperature, and she died 23 days following her admission to the hospital. An autopsy was obtained. The peritoneal cavity contained one liter of straw colored fluid and each pleural cavity approximately 1,200 cubic centimeters. There was a small pericardial effusion (340 c.cm.) and there was an extensive and obviously important degeneration of the right ventricular muscle on the basis of vascular disease. This involvement of the right ventricle appeared to account for the congestive phenomena. The pericardial effusion probably accounted for the reduced pulsation. The combination of pure ventricular failure with a small amount of pericardial fluid was mistaken by us for constrictive pericarditis.

3. Extracardiac disease. (a) Portal cirrhosis. There should be little difficulty in differentiating constrictive pericarditis from portal cirrhosis of the liver. The possibility of confusion is illustrated by the fact that a number of patients with constrictive pericarditis have had an omentopexy, usually without benefit. The absence of a history suggestive of cirrhosis of the liver, the presence of a paradoxical pulse, the elevated pressure in the neck and arm veins, and the decreased pulsations of the heart should serve to make the diagnosis of chronic constrictive pericarditis. It is well to remember that both diseases may be present.

An example of chronic constrictive pericarditis, which had been diagnosed erroneously as cirrhosis of the liver, is presented in detail. An omentopexy had been performed in an effort to improve his condition.

E. E., a white male farmer of 21 years, entered the Vanderbilt University Hospital on August 15, 1934, complaining of swelling of the abdomen and shortness of breath.

On December 28, 1929, 5 years before admission, this patient began to ache throughout his body. His physician said that he had either influenza or pneumonia. He went to bed and remained there 3 days before returning to work. Approximately 2 weeks after returning to work, the patient began to be short of breath. A diagnosis of empyema was made; a resection of two ribs on the left side was performed, and pus was encountered in the pleural cavity. He was in the hospital for 9 days and upon getting out felt quite well for 2 or 3 weeks. Shortly thereafter he noticed swelling in sundry parts of his body in the following order: scrotum, legs and thighs, abdomen, arms, face and eyes. The swelling of the face was quite marked in the morning but disappeared during the day. Coincident with the generalized edema he became more short of breath. He did not feel strong enough to return to work. In April, 1930, he was given diuretics which reduced the amount of edema, and he felt much better for about 5 months when his abdomen began to enlarge again. In the summer of 1931, he went to a hospital where he remained for 47 days. Three abdominal paracenteses were performed and 13, 8, and 9 liters of fluid were removed from his abdomen. An omentopexy was performed during this stay in the hospital. Following his discharge, he got along fairly well until the winter of 1933 when he began having mild chills with fever at intervals. Several times he coughed up small amounts of blood. In March, 1934, while loading hay, he was struck on the left side of the abdomen by a hay fork which he was using. After this blow his abdomen was tender to pressure, and began to increase gradually in size. On April 26, 1934, he again entered another hospital for a period of 19 days. Fluid was withdrawn from his right chest at that time. Following this, it was necessary for him to return to the hospital on several occasions and on each visit fluid was withdrawn from the chest. The patient had been at home for 6 weeks previous to his admission to the Vanderbilt University Hospital.

On examination he was found to be a poorly nourished white male who did not appear acutely ill. He was slightly dyspneic while lying flat in bed. There was marked engorgement of the neck veins when he was lying flat in bed, but this prominence of the veins disappeared when he sat up. There was marked fullness of the lower half of chest, with definite increase in the anteroposterior diameter. Expansion was

limited on both sides, and this limitation was most marked on the right. There was dullness (in some areas flatness) over the entire right chest, with markedly diminished breath sounds. The left chest was resonant throughout except for dullness in the lower half of the back. There were diminished breath sounds in this area. The heart sounds were distant; there were no murmurs. There was no shifting of the cardiac dullness with change in position. The veins of the chest wall were unusually prominent.

The abdomen was distended. There was a definite fluid wave and shifting dullness was present. The liver was felt 6 centimeters below the right costal margin. The edge of the liver was firm and smooth. There was a hernia at the lower part of an abdominal scar.

The temperature was 99 degrees F., the pulse rate 120 per minute, respirations 21, and the arterial blood pressure was 90/60 millimeters mercury.

The x-ray report at the time of this admission was as follows: "There has been a resection of the left ninth rib posteriorly. Slight thickening of the pleura in the left costophrenic angle. Practically complete obliteration of the right lung field due to fluid and thickened pleura. The right border of the heart is not made out. Practically no pulsations seen in the cardiac shadow on the left side. No activity seen in the left lung."

Electrocardiographic study revealed notched QRS in all leads and no shift of axis.

The patient was discharged from the hospital September 2, 1934, and readmitted September 24, 1934. During this time his condition had remained practically the same. The venous pressure at the time of second admission was 195 millimeters water. The circulation time, arm to carotid, was 52 seconds by the sodium cyanide method. After further observation it was decided that this patient should have a decortication of the heart.

Operation was performed October 4, 1934. A left parasternal incision was made and the third, fourth, and fifth costal cartilages, together with parts of the ribs, were removed. There were many dilated veins beneath the cartilages. No cardiac pulsations were visible when the pericardium was exposed. Incision was made through a thickened scar which was 7 to 10 millimeters in thickness. Good pulsation of the heart was noted after a part of the scar had been removed. An area of scar approximately 6 by 6 centimeters was cut away. A great deal more of the scar which was not removed was freed from the heart. Good pulsations resulted. The wound was closed without drainage. Microscopic sections showed a thickened, fibrous membrane which was quite vascular on its outer surface.

Following the operation, the patient made a slow but progressive improvement. He returned to the hospital approximately 1 year following the pericardectomy for repair of the hernia at the site of the omentopexy. He has been performing hard manual labor for the past 5 years. Normal pulsations of the heart are visible under the fluoroscope.

b. Mediastinal tumors. A tumor which is located in such position that it compresses the superior and inferior vena cavae may be associated with findings which are quite similar to those caused by constrictive pericarditis. There is less apt to be a general suppression of the pulsations of the heart in the patient with a mediastinal tumor, and more apt to be a greater venous pressure in one caval area than in the other.

c. Nutritional edema. The differentiation between edema due to constrictive pericarditis and nutritional, which it may simulate, depends primarily on the determination of the venous pressure and the concentration of serum proteins. The former is elevated in constrictive pericarditis and normal in uncomplicated nutritional and nephrotic edema. The serum proteins (and colloid osmotic pressure) are lowered in nephrosis and nutritional edema and normal or only slightly lowered in constrictive pericarditis. Nutritional edema, however, occurs as a complication of many other diseases, including chronic heart disease, and hence may add to the edema of constrictive pericarditis, the lowered serum proteins augmenting the edema-producing effect of the increased venous pressure. Under these conditions the relative importance of the nutritional factor may be judged from the level of the serum proteins or the colloid osmotic pressure.

d. Multiple thrombosis of veins. A patient with multiple thrombosis of veins has been observed who presented a picture highly suggestive of chronic constrictive pericarditis except that the pressure in the tributaries of the superior vena cava was slightly lower than that usually found in the latter condition. A description of this case follows.

O. A. A., colored, aged 19 years, entered the Vanderbilt University Hospital January 2, 1936, with a complaint of "swollen sore stomach and headaches." Slight weakness and shortness of breath was observed 2 years previously. Seven months prior to admission, he noted enlargement of the abdomen and a mass was felt extending below the costal margin. The mass was somewhat tender to pressure. In addition, he had had some pain of both the right and left sides of the chest. The findings on examination included prominent veins of the abdomen and chest, a greatly enlarged liver and spleen, and dullness in the flanks. The arterial blood pressure was 155/130. The venous pressure in an arm vein was 125 milli-

meters water. The red blood cell count was 7,000,000 and the hemoglobin 16 grams. Fluoroscopic examination of the heart showed definite but small pulsations.

An exploratory laparotomy was performed: and the veins of the omentum were found to be tremendously enlarged and distended. The liver was greatly enlarged and quite dark in color. The spleen was enlarged and hard. There was a moderate amount of free peritoneal fluid. The impression was that there was partial obstruction of the hepatic veins. The patient died subsequently and an autopsy was performed. There was a definite constriction of the inferior vena cava as it passed through the groove in the liver. At this point the circumference of the vena cava was only 2.5 centimeters whereas its circumference was 7.5 centimeters at the level of the entrance of the renal veins. The constricted area was 8 centimeters long. The wall of the vein was thickened, apparently due to fibrous tissue. The hepatic vein orifices were smaller than normal and several contained thrombi. In addition, there was thrombosis of the splenic vein. The pericardium and heart were normal. A photograph of the veins is shown in Figure 6.

e. Conditions associated with a marked increase in the intrathoracic pressure may be confusing and occasionally there may be difficulty in differentiating diseases such as chronic asthma from constrictive pericarditis. In these conditions the heart is usually small and the venous pressure is elevated. A paradoxical pulse may be present. Differentiation may be made on the basis of signs of pulmonary disease.

INDICATIONS FOR AND TIME OF OPERATION

It is generally agreed that an acute pyogenic pericarditis should be drained and that an acute tuberculous pericarditis with a large accumulation of fluid should be treated by aspiration rather than by open drainage. It is accepted that when constriction is associated with a pyogenic infection, an appropriate amount of thickened pericardium should be removed, and the infected area should be drained. It is generally agreed that a pericardectomy should be performed when a patient has constrictive pericarditis of marked degree due to a process, tuberculous or otherwise in origin, which is healed or inactive. If the disability is only partial and is not progressing, operation may be postponed with the hope that it will not be necessary. If one is convinced that an operation will be necessary, there is danger in postponing it after the chronic stage of the disease has been reached. Delay results in greater atrophy of the myocardium and the dangers of entering the heart cavities during the decortication and of dilatation of the heart at and subsequent to the operation are increased. In other words, pericardectomy is indicated when the active process has been replaced by a healed scar and before marked cardiac muscular atrophy occurs.

It is obvious that the patient does not always present himself at this ideal time. There is a variation in the time required for an active tuberculous infection to become healed. It may be stated in general that an interval of at least 6 months and preferably a longer time should separate the initial infection and the removal of the scar. As will be pointed out subsequently, however, the unfavorable course of the patient may render imperative an earlier operation.

There is a difference of opinion as to the attitude which should be adopted when the constriction is caused by a thickened pericardium and epicardium in which a tuberculous infection is still present and when the patient's condition is becoming progressively worse. A frequent occurrence in tuberculous pericarditis is the development of increased signs of systemic congestion as the fluid disappears from the pericardium and as the heart-pericardium shadow becomes smaller. The patient who has constriction and an active tuberculous infection has both as causes for his signs and symptoms, and the treatment is unusually complicated. Most of the patients, studied and operated upon by us, have had activity in the sense of demonstrable tubercles in the pericardium and epicardium but have not had proliferative activity with fluid. The stage of the disease that has been present in most of our patients at the time of operation is demonstrated in Figure 7 and the more active proliferative stage in which operation should be avoided, if possible, is shown in Figure 8.

Volhard expressed the opinion that the proliferative stage of tuberculous pericarditis is not well suited for operation. Churchill (24) stated:

"Active tuberculosis of the pericardium may produce the entire syndrome of chronic constrictive pericarditis. The point at issue is whether operation can be effective if performed during the active phase of the infection. The reports in the literature of operations performed during this period are uniformly discouraging and confirm our personal experience."

The authors agree entirely that the operative procedure is easier and that the mortality rate is lower if operation can be postponed until the tuberculous process has become inactive. Such has not always been possible in our experience because of the unfavorable course of the disease. The general policy which has been adopted is that the operation should be deferred during the active stage as long as the patient's condition is stationary or is improving. If, however, the patient is losing ground and the venous pressure is rising despite the fact that the heart-pericardium shadow is decreasing, indicating the disappearance of fluid, operation should be proceeded with even though some activity is still present. Even in the presence of proliferative activity without fluid, operation may be practicable and the decision for or against it should be made on the basis of the severity of the interference with the circulation rather than on the basis of the presence or absence of activity of the tuberculous process. One has to evaluate the relative rôles of constriction and of infection. If constriction is responsible for most of the symptoms and signs, operation is indicated; if infection is responsible for the predominating features of the illness, nonoperative treatment is to be preferred. If both infection and obstruction are important in producing the disability, one should attempt to make the difficult decision as to therapy after balancing the advantages and disadvantages of operative and nonoperative treatment.

This discussion would be incomplete without a consideration of the treatment of patients in whom the obstruction is only moderately severe. Since a moderate amount of danger in association with pericardectomy is inescapable, nonoperative treatment is usually advisable if the disease is not progressive and if the disability is not severe. Under these circumstances, measures such as the restriction of fluid and salt intake, the use of diuretics and the aspiration of serous cavities, if indicated, will usually add to the comfort and reduce the incapacity of patients. Digitalis is not effective save in the rare cases in which there is heart failure as well as obstruction.

In summary, one should realize in considering the indications for and the time of operation that successful therapy is dependent upon the ability of the surgeon to remove enough scar to release the heart, upon the capacity of the weakened heart muscle to perform its work, and upon the nonprogression of the inciting disease. The decision as to therapy is in many instances a difficult one to make.

THE OPERATION

Preoperative treatment. After it has been decided that pericardectomy is indicated, the patient should be got into the best possible condition. This consists in the main of reducing to a minimum the results of the pericardial constriction, that is, the edema, ascites, and pleural effusion. This part of the treatment is similar in most respects to that of heart failure. The patients should be placed on a low salt diet (approximately 2.0 grams daily) and the fluid intake should be limited to approximately 1,500 cubic centimeters daily. If the blood studies show a reduction of plasma proteins, which is a frequent finding, a high protein diet should be given. Large accumulations of fluid in the pleural or peritoneal cavities should be removed by aspiration. Any one of a number of diuretics may be used in an attempt to cause the disappearance of edema. There is a variation in the individual response to these agents. The advice of Heuer and Stewart is as follows:

"Of the drugs used to mobilize fluids, mercupurin has been found to be the most satisfactory. It is usually given in 2.0 cubic centimeter doses intravenously at 3 day intervals. To some patients ammonium chloride, 3 grams per day, was given at the same time to enhance the diuretic effect. Theocalcin, 4.5 grams daily, urea, 30 cubic centimeters of a 50 per cent solution, twice daily and aminophyllin 0.1 gram 3 times per day were all tried but with less effect."

Regardless of the agent used, the patient should be rendered free of as much excess fluid as possible. It is particularly important that large accumulations of fluid in the pleural

cavities should be removed prior to the anesthetic and operation.

An interesting question is concerned with whether or not patients with constrictive pericarditis should be digitalized, and particularly so when operation is contemplated. Harrison and Leonard have shown that digitalis decreases the output of the heart of normal dogs. Burwell, Neighbors, and Regen (20) found that it decreases the cardiac output in normal men. Burwell and Strayhorn (21) found in a patient with constrictive pericarditis that digitalis decreased the cardiac output per minute due chiefly to a decrease in rate with little change in the output per beat. Harvey and Whitehill state:

"Inasmuch as these patients seem abnormally sensitive to digitalis and develop cardiac arrhythmias with small doses, and since the heart failure is usually due to cardiac tamponade, the drug, generally, is not indicated. In those patients who continue in failure after the pericardial fluid has been removed, particularly the older ones with arteriosclerosis, it might be useful."

Burwell (16) states that there are 3 important exceptions to the general rule of the nonusefulness of digitalis in these patients: the occasional patient who has auricular fibrillation with tachycardia in addition to pericardial obstruction; the patient about to be subjected to pericardectomy whose possibly atrophied myocardium may have imposed upon it increased demands if the operation relieves the obstruction to inflow; and the patient who has both obstruction and myocardial insufficiency.

Preanesthetic sedatives should be given in moderate amounts but not to the extent that the respirations are depressed. Compatible blood should be available for transfusion in case such is found to be necessary as a result of copious bleeding during the operation. Fluids, including blood, should not be introduced intravenously unless absolutely necessary.

The anesthetic. There is no unanimity of opinion as to the most desirable anesthetic agent for this operation. Most surgeons are agreed that general anesthesia is indicated because of the magnitude of the procedure and of the possibility of the accidental injury of the heart muscle and of opening one or both pleural cavities. It is because of the latter possibility that Churchill (24) uses intratracheal anesthesia, the gas-oxygen-ether sequence being employed. Of the 7 patients subjected to pericardectomy by Heuer, 4 were anesthetized by the open drop ether method, 2 with drop ether administered through an intratracheal tube (Magill), and ethylene-oxygen-ether mixture was used in the remaining patient. Harrington used cyclopropane as the anesthetic agent in the 9 patients on whom pericardectomy was performed. This was administered through a tightly fitting mask but he cautions that an intratracheal tube should be available for use if necessary. In the author's experience, the agents which have been used include local alone, local combined with nitrous oxide and oxygen, with cyclopropane and oxygen and with gas-oxygen-ether. Our routine at present consists of local infiltration for the incision and removal of bony structures followed by the gas-oxygen-ether sequence during the time that the pericardial scar is being removed. Usually an intratracheal tube is not employed. The most unsuitable agent that we have used is nitrous oxide. It is impossible to obtain satisfactory anesthesia without causing anoxia. There is a pre-existing impairment of the circulation of the heart muscle as well as structures elsewhere and it is unwise to cause additional anoxia.

The impression exists, without definite proof, that cyclopropane may exert toxic effects on the heart. Robbins and Baxter (44) found that the cardiac irregularities which develop at the time of, or soon after, respiratory arrest as caused by cyclopropane are not due to the anesthetic agent itself but to anoxemia. They noted further (45) that cardiac irregularities which are observed in dogs under cyclopropane anesthesia after premedication with morphine may be abolished by barbital or amytal. In view of these findings, it appears that a barbiturate rather than morphine should be employed when cyclopropane is to be used. As stated previously, it is our impression that gas-oxygen-ether is preferable to oxygen-cyclopropane in operations on the heart.

Beck and his associates (3, 6) found in animals that exposure of the heart and great vessels to atmospheric air pressure results in a significant reduction in the cardiac output.

As a result of these observations, they recommended the revival of the use of the Sauerbruch negative pressure chamber. One of us (A. B.) was unable to confirm the experimental observations under slightly modified conditions (10). At any rate, a diametrically opposite viewpoint to that of Beck is expressed by Churchill (24). He states:

"While Beck focuses his attention upon failure of the peripheral circulation from a reduced filling of the heart, I consider the real hazard of the operation to lie in exposing the weakened musculature of the heart after it has been released from the scar to too great a venous return. From this point of view the effect of atmospheric pressure on the surface of the heart would be welcomed. I have deliberately attempted to reduce the venous return to the heart during and immediately following the operation."

Measures which may be used to reduce the venous return, include the upright position and the reduction of the blood volume by bleeding. However, as Churchill has emphasized, it must be remembered that a reduction in the effective venous pressure may be dangerous if the heart is not relieved of its constricting scar.

Position of patient. The patient should be placed on the operating table on his back in either the recumbent or semirecumbent position. Churchill (24) and Schmieden (48) prefer a slightly upright position, using a dental chair, in order to reduce somewhat the venous return to the heart. There is no strong objection to the recumbent position unless the patient is dyspneic.

Exposure of pericardium. Most surgeons are agreed that the operation should be performed through an extrapleural approach and that the incision should be made to the left of the sternum. Median sternotomy is not necessary as an adequate exposure can be obtained through a left parasternal incision. The incision should be such that the heart can be exposed from base to apex. The midportion of the incision should be over the sternum or at its left border and the lateral projections of the incision should overlie the uppermost and lowermost costal cartilages which are to be resected, extending to the midclavicular line. The pectoralis major muscle is freed from the sternal border and is reflected laterally with the skin flap. It is usually advisable to remove the third, fourth, fifth, and sixth costal cartilages and short segments of the adjoining ribs. There is a difference of opinion as to whether the perichondrium and periosteum should be included with the cartilages and ribs. Both Heuer and Harrington state that it is not necessary to include them. The present authors agree with Schmieden (48) and with Churchill (24) that it is desirable to have a flexible precordial area over the liberated heart and that the perichondrium should be removed in order to avoid regeneration. Furthermore, it is our impression that one is less apt to have a chronic discharging sinus when the process is tuberculous in origin if perichondrium is removed. In this connection, it is quite important that all cartilage which is damaged should be removed. The intercostal muscles are divided at the left sternal border, and the internal mammary artery and vein are usually ligated at the upper and lower extremes of the incision. The thin layer of muscle and connective tissue overlying the pericardium is divided, and the left half of the sternum is removed with rongeurs or the Lebsche sternum knife.

Beginning at the left sternal border near the lower end of the incision, the left pleura is dissected laterally for a considerable distance, preferably to the left phrenic nerve. It is well to remember that there may be no so-called "area of safety" and that the right and left pleural reflections may be in contact with each other. In most instances, the greater part of the dissection of the pleura may be carried out by blunt dissection, a thin layer of gauze applied to a finger being used. The experience of most surgeons is that an opening is made in the pleura during the dissection in about one third of the operations. This usually causes no embarrassment to the respirations and it is unnecessary in most instances to attempt closure of the tear in the pleura. As will be commented upon subsequently, an additional reason for not closing a pleural opening is that there are some points in favor of drainage of the decorticated area into the pleural cavity unless pyogenic organisms are present. It is our policy to delay dissection of the right pleural reflection until after removal of the pericardial scar has been begun.

In the sickest patients, particularly the elderly, Bigger (36) has suggested that the operation should be performed in stages. At the first stage, the rigid structures of the chest wall may be resected and a small portion of the scar near the left border of the heart may be removed. At the second stage, the further removal of scar may be carried out through the same incision or through a corresponding incision to the right of the sternum.

Removal of scar. Before the technical features concerned with the removal of the scar are considered, points of importance in connection with the choice of site for the beginning of the decortication and also the extent of the decortication will be considered. It should be borne in mind in this discussion that it is impossible before beginning the removal of scar to distinguish the various parts of the heart. The left ventricle lies almost entirely laterally and posteriorly, and the portion of the heart in contact with the anterior chest wall is largely the right ventricle. The most frequent mistake is the assumption that one is decorticating the left ventricle when in reality it is the right one. First, the site for the initial removal of the scar will be discussed. It is generally believed that it is important to liberate part of the left ventricle from the scar before decorticating the right ventricle. The reasons for this are two in number. The muscle of the left ventricle is thicker than that of the right, and there is less danger on the left of entering the cavity of the heart when one is attempting to establish a line of cleavage between the heart muscle and the scar. The second reason for freeing the left ventricle first is the theoretical danger of pulmonary edema. Initial release of the right ventricle permits a greater flow of blood into it and thence into the lungs. Since the transmission of blood through the lungs has not been facilitated by decortication of the left ventricle there may result an increase in the amount of blood in the lungs. This in time may lead either to pulmonary edema or to dilatation and failure of the right ventricle. This reasoning appears to be sound but in actual practice we have seen no harmful effects in those instances in which the right ventricle has been decorticated before the left. As stated previously, it is our impression that the surface of the right ventricle is often believed to be that of the left one.

The second point has to do with the extent to which the decortication should be performed. There is some difference of opinion on this subject. Most are agreed that actual decortication of the auricles is a hazardous procedure due to their thin walls and should not be attempted. If the ventricles are properly freed of scar, the undecorticated auricles are able to conduct sufficient blood to the ventricles to supply the needs of the body. Churchill (24) states that free motion of the right auriculoventricular groove is an important physiological mechanism in the action of the right heart and that every effort should be made to free this area of scar. Several observers have commented on the need for the removal of the scar from the site at which the vena cavae enter the heart. It is our impression that this is rarely necessary and even more rarely accomplished successfully. The extent of the removal of scar as advocated by Heuer is as follows:

> "We have removed as completely as possible the pericardium over the left and right ventricles, but have not attempted to remove the adherent pericardium over the auricles. An effort has been made to free the apex, and if possible the dissection is carried well down on the diaphragmatic portion of the pericardium. No attempt has been made to go beyond the right auriculoventricular groove; nor have we attempted to free the great vessels at the base."

Schmieden (48) states that complete liberation of the left ventricle is the most important part of the procedure and that in most cases this is the only surgical measure that is necessary. We question seriously the correction of this view. Most patients with constrictive pericarditis have remarkably little dyspnea and edema of the lungs in comparison with the marked evidence of congestion in the systemic circulation. Most of the signs and symptoms are attributable to back pressure from the right side of the heart. In our experience, major emphasis has been devoted to decorticating the right ventricle. In one patient who has obtained a good result, all or certainly most of the decortication was performed on the right, since bleeding from an unknown source had been encountered during

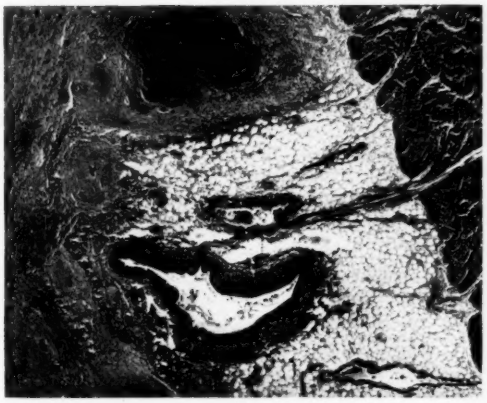

Fig. 9. Section showing an active tuberculous infection of scar. Note the large artery which is intimately attached to the scar. Autopsy specimen. ×20.

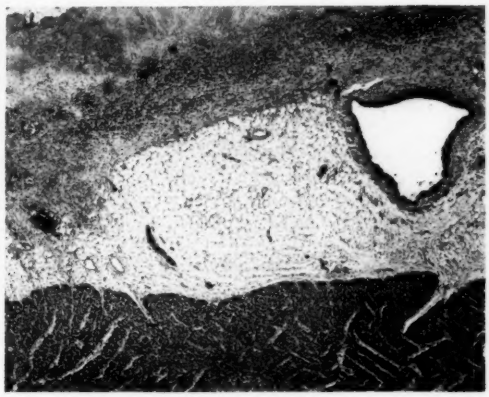

Fig. 10. Section showing caseation and giant cells. Note the large blood vessel which is anchored more firmly to the scar than to the heart muscle. Autopsy specimen. ×16.

the attempt to remove scar from the left ventricle.

We shall return now to a description of the operative procedure itself. After having obtained an exposure of the pericardium which extends to or almost to the left phrenic nerve, an incision is made into the scar overlying the left ventricle. This is probably the most tedious part of the operation. Much of the ease and success of the operation is dependent upon finding the correct plane of cleavage. It should be remembered that it does little if any good to remove the scarred pericardium if thickened fibrous epicardium is left behind. The plane of cleavage should lie preferably between the layer of organized exudate on the exterior of the epicardium and the epicardium itself. It should not lie immediately next to or in the heart muscle. As is well known, the major coronary arteries are located quite superficially. If one is unable to establish the correct plane of cleavage, the point of attack should be shifted. Usually slight motion of the heart can be seen when the incision in the scar has been extended to the correct depth. The incised edges of the scar are then grasped with instruments, and the decortication is extended by sharp and blunt dissection. A sharp instrument should be used for the division of all strands of scar tissue which extend into the heart muscle. One may encounter areas in which there are unusually dense adhesions and the cleavage plane is lost. As Churchill (24) has stated, this is particularly apt to be encountered in the region of the left descending coronary artery. This area should be approached with caution in order to avoid injury to this important vessel. If one is unable to be certain as to the relationship of the scar to the vessel, dissection at that point should be abandoned temporarily and a cleavage plane further to the right should be found.

As the sternum is approached, it is usually easy to push the right pleural reflection laterally with gauze if the heart is dislocated slightly to the left by making traction on the scar. One should pause frequently during the dissection of the scar in order to allow the heart to regain its normal rhythm. Bigger (8) has pointed out the importance of not excising the scar as it is freed and of leaving temporarily a flap of the tissue at the point of the dissection in order that it may be used for covering a rent in the heart muscle if this unfortunate accident occurs. As has been stated, the removal of the scar should be extended, if it is feasible, to the region of the phrenic nerve on the left, to the neighborhood of the inferior vena cava on the right, and the apex of the heart should be liberated. The scar that is removed usually measures approximately 8 by 10 centimeters, and it may vary in thickness from 1 millimeter to more than 1 centimeter. It is very gratifying to note that the

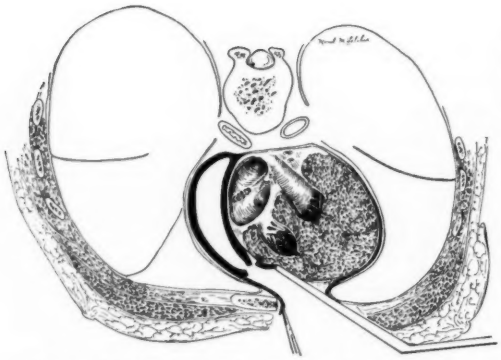

Fig. 11. Method for developing a new line of cleavage to separate the inner wall of cavity containing fluid from the surface of the heart. (From Churchill, E. D., *Ann. Surg.*, 1936, 104: 522.)

removal of a relatively small area of scar tissue will result in the freeing of a rather large part of the surface of the heart.

There are several points which deserve additional comment. As has been stated, calcification of the pericardium may or may not be present. If present, there may be a complete bony shell. The calcified tissue may extend into the heart muscle, and in such instances one is particularly likely to make an opening into a cavity of the heart. One should not hesitate to leave isolated areas of fibrous tissue or calcium if the danger of damaging the heart wall in the dissection appears to be great.

As has been stated, the large coronary vessels have a superficial position and are apt to be injured in the process of removing the scar. Sections of several autopsy specimens show that these vessels may become more intimately attached to the scar tissue than to the underlying heart muscle. Examples of this are shown in Figures 9 and 10. The death of one of our patients probably resulted from including a large branch of a coronary artery with the scar that was removed. Autopsy revealed rupture of the heart at the site of necrosis of part of the wall of the right ventricle. As has been pointed out, Churchill (24) recommends extreme caution when approaching the sulcus formed by the left descending coronary artery. This may be approached from both sides after a plane of cleavage to the right and left of it has been established.

In an occasional case in which the major part of the pericardial cavity is obliterated, one may find an encapsulated area of fluid, both the pericardium and epicardium being thickened. Churchill (24) has emphasized the importance of removing the wall of the cavity that is in contact with the surface of the heart. Little good results simply from removing the wall that corresponds with the parietal pericardium. The point at which the line of cleavage should be developed is shown in Figure 11 (from Churchill).

If the right ventricle cannot be decorticated properly at the primary operation through the left parasternal incision, this can be attempted subsequently through a similar incision on the right. This has not been necessary in our experience. If the apex of heart cannot be liberated, Schmieden (48) states that there is no objection to the inclusion of the left phrenic nerve with the scar that is removed, thereby resulting in a paralysis of the left side of the diaphragm.

As has been said, the regular rhythm of the heart may be disturbed by the operative procedure. This is particularly likely to occur when traction is made on the scar. Whether or not an irregularity occurs, it is important to allow frequent "rest" periods. During this time, warm moist sponges should be applied to the surface of the heart. Heuer and Stewart have described accurately the alterations in the heart beat in their patients.

"In the 7 patients subjected to operation, the heart beat, as counted at the wrist, varied between 100 and 160 and in all cases was over 130 during the greater part of the operation. In 2 cases the heart action was fairly regular; in 5 markedly irregular. In some cases there were periods of paroxysmal tachycardia; in others transient ventricular fibrillation, in still others such complete loss of rhythm that the cardiac action could be described only as complete arrhythmia. Periods of transient stoppage of the heart occurred not infrequently."

Beck and Mautz (7) recommend the direct application of procaine or metycaine to the heart for the reduction of irritability of the myocardium. There was no effect on the cardiac action in the 1 case in which this method was used by Heuer. Frequent interruptions, allowing the heart to rest, appear to be of the greatest aid in reducing irregularities.

Fig. 12. Showing postoperative photograph taken on February 2, 1937 of patients listed as Cases 1, 2, and 5 in Table II. Patient on left had a healed pericardial scar, subsequently developed tuberculous pericarditis from which he recovered. Operation by Dr. Bigger, second successful case in America. Patient in center had constrictive pericarditis following staphylococcus infection. Organisms still present at time of pericardectomy. Patient on right had tuberculous constrictive pericarditis with activity. This is the only patient on whom we have performed a second operation for the removal of additional scar. The venous pressure is essentially normal in these three patients and fluid accumulation has ceased. Photomicrographs of the scars are shown in the succeeding three figures.

If an opening has been made unintentionally into the left pleura, it is probably just as well to make no attempt to close it. In fact, Griswold recommends that an opening be made deliberately into the left pleural cavity in order that any fluid which forms about the heart may accumulate there, from which site it may be aspirated. This would seem to be unwise in the presence of an active infection and should be used only when there is a healed scar.

Closure of the incision. After the decortication has been completed, the skin and muscle flap is replaced over the defect, and the incision is closed. The authors agree with Churchill and Heuer that the insertion of a drain is usually unnecessary and unwise. If there is evidence of an active residual pyogenic infection, a small drain should be introduced.

The danger of tamponade from an accumulation of fluid in the precordial region is not great, since the fluid can pass into the tissue spaces of the chest wall. It is not as though it accumulated beneath a dense, inelastic scar. If the accumulation of fluid should embarrass the heart's action, it can be aspirated usually without difficulty.

Postoperative treatment. A number of surgeons, including Churchill and Harrington, state that all patients following pericardectomy, should be placed in an oxygen tent or cabinet. We agree with Heuer that the inhalation of a high concentration is usually unnecessary unless indications such as dyspnea and cyanosis are present. Blood transfusions should be avoided if possible because of the danger of cardiac dilatation. If blood or other fluids are given intravenously, not more than 30 cubic centimeters per minute should be introduced.

Postoperative treatment consists in the main of a continuance of measures which were used before operation, such as limitation of fluid and salt intake and the use of diuretics when indicated. The accumulation of fluid in

Fig. 13. Section of pericardium showing a thickened and hyalinized scar without evidence as to etiological agent. Tuberculous peritonitis demonstrated at subsequent operation. ×10.

serous cavities usually does not stop abruptly and aspiration should be performed when indicated.

RESULTS—PROGNOSIS WITH AND WITHOUT OPERATION

Churchill (23) in 1929 collected the reported operations or attempts at operation on patients with constrictive pericarditis. The total number of cases was 37 and the results were as follows: (1) The operation was not completed in 5 patients; (2) there were 7 deaths directly attributable to the operative procedure; (3) there was no improvement in 2 patients who survived the operative procedure; (4) the improvement was transitory in 4 patients who subsequently died of the original disease; and (5) the result was excellent in 19 patients, or 59 per cent of the total.

Fig. 15. Case 5. Section showing a thickened scar due to proliferative tuberculous infection. Caseation and giant cells are to be observed. ×19.

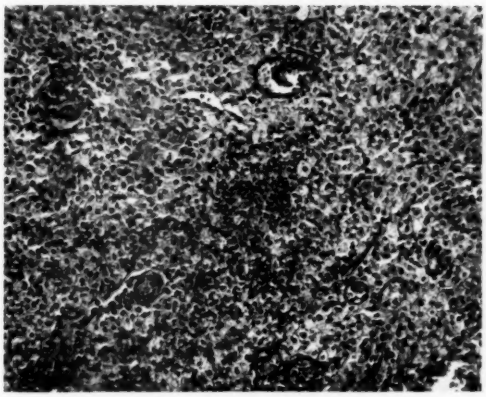

Fig. 14. Section showing chronic inflammation due to staphylococcus aureus. ×125.

A similar survey of the literature was made by Heuer and Stewart in 1939. They found the reports of 143 patients in whom the pericardectomy had been performed for chronic constrictive pericarditis. Of the 143 patients, 50, or 35 per cent, were reported as cured and 25, or 17.5 per cent, as improved. Nineteen patients died while on the operating table and 28 died in the immediate postoperative period, a total of 47 patients, or 32.8 per cent of the entire number. Thirteen patients died at variable times after discharge from the hospital, 6 of these lived for more than a year and were probably improved by the operative procedure. The 7 remaining patients in this group of 13 died from 2 to 8 months following the operation, probably as a result of the continuation of the disease. The results of surgical therapy were not obtainable in 8 of the cases. Heuer and Stewart state: "The final analysis, then, shows that of 135 patients in the reports of whom data are available, 50, or 37 per cent, are cured; 31, or 23 per cent, improved; and 54, or 40 per cent, died, either during or soon after operation." Heuer and Stewart make the additional comment that it should be realized that the 150 cases represent the sum of the experience of 49 observers, 27 of whom have operated upon only a single patient, 7 of whom have operated upon 2 patients, 6 have performed the operation 3 times each, and only 8 surgeons have operated upon 5 or more patients.

The total number of instances in which pericardectomy has been performed is probably at present considerably larger than the 143 cases analyzed by Heuer and Stewart. There are undoubtedly a number of cases that have not been reported. Pericardectomy for constrictive pericarditis has been performed on 20 patients in the Vanderbilt Hospital and probably on a slightly greater number of patients in both the Massachusetts General and Lakeside Hospitals, some of which are unreported. Heuer of Cornell reported last year the results of 7 such operations and Harrington of the Mayo Clinic recently described the results of 9. The previous findings that approximately 60 per cent of the patients are either cured or greatly improved by the operation is corroborated by recent results. Heuer's series is unique in that there have been 7 operations without a death. Three of the patients are cured and 4 are markedly improved.

As in all types of surgery, the mortality rate and the percentage of cures is dependent somewhat upon the etiological agent and the stage of the disease. For example, a comparison of the mortality rate in operations on healed pericardial scars with that in operations on tuberculous processes which are still active is somewhat like comparing the mortality rates in appendicitis with and without peritoneal infection. It is likely that the percentage of disappointing results will always be rather high if pericardectomy is performed on patients with tuberculous constrictive pericarditis without fluid but with activity. The important question is whether pericardectomy in this stage of the disease results in more cures than nonoperative treatment. It should be emphasized that we refer to the stage in which the fluid has disappeared, the venous pressure is rising, the patient is losing ground, and activity is still present. Particular attention is devoted to this aspect of the subject because there is general agreement that pericardectomy is indicated in all instances in which there is a healed scar that is causing incapacitation.

The mortality rate in tuberculous pericarditis, acute or chronic, is extremely high. Harvey and Whitehill analyzed the results in the Johns Hopkins Hospital, and they state: "Of 20 proven cases with effusion 16, or 80 per cent, died, and 15 of the 17 proven cases without an effusion succumbed, making a total mortality of 83 per cent." Keefer reviewed a series of 20 patients with tuberculous pericarditis at the Boston City Hospital. Of this group, 18 died and 2 survived. The findings of others support these observations. The picture, however, is not as grave as it appears to be. There are undoubtedly a good many instances of mild tuberculous pericarditis in which recovery occurs. A positive diagnosis can be made only by aspiration (with demonstration of organisms), by operation, or by autopsy. Tubercle bacilli cannot be demonstrated in the pericardial fluid in a good many instances of pericarditis which are almost certainly tuberculous in origin. These are the less severe cases in which spontaneous recovery is apt to occur. That a severe infection may result in a healed scar is suggested by this finding in patients with other tuberculous lesions. Granting that these conjectures are correct, the prognosis is extremely poor in a patient in whom tubercle bacilli can be demonstrated in the pericardial fluid. If the acute infection is survived, there results in nearly all instances (13) a constricting mass of tuberculous tissue which may be converted subsequently into a dense scar.

An analysis of the 20 patients in the Vanderbilt Hospital on whom pericardectomy was performed shows that the scar was proved to be tuberculous in origin in 13, that it resulted from a previous staphylococcus infection in 3 and that the etiological agent was undetermined in 4. One of the latter 4 subsequently entered the hospital with tuberculous peritonitis, and the assumption appears warranted that the pericardial scar was tuberculous in origin. The etiological agent could not be determined in a second of these patients because hemorrhage necessitated the abandonment of the operation before any scar was removed. The patient developed pneumonia from which she died, and an autopsy was not performed. Of the 13 patients who had an active tuberculous process in the scar at the time of operation, 7 are living and 6 are dead. Of the 10 who survived the operation for more

than 3 days, 7 are living and 3 are dead. It seems only fair to state that these 3 early deaths were due to the operation and not to the fact that activity was present. Two of the patients in Churchill's series (24) had an active tuberculous pericarditis at the time of operation. He stated: "Both patients died promptly, one on the operating table and the other a few hours subsequently, their lives being shortened a few days as a result of the operative procedure." Again, it is possible that death would have occurred even if the process had been inactive. The 3 late deaths in the Vanderbilt series following pericardectomy for a tuberculous scar occurred 2 months, 6 months, and 2 years following operation and were due to miliary tuberculosis. There is no reason to believe that their lives were shortened by the operation. Harvey and Whitehill found that the average duration of life from the onset of symptoms in 15 patients with tuberculous pericarditis without effusion was approximately 5 months. Pericardectomy was not performed on any of these patients and the diagnosis was confirmed at autopsy. The average duration of the disease in Keefer's 18 fatal cases was approximately 3 months. There is no doubt but that it is better to operate upon constrictive pericarditis due to tuberculosis in the healed rather than in the active stage but our findings indicate that the prognosis is far from being hopeless when it is necessary to operate before all of the activity has subsided.

Three of the 20 patients on whom pericardectomy was performed or attempted had had acute pericarditis due to the staphylococcus some months previously. There were small residual abscess pockets containing staphylococci at the time of pericardectomy. Two of the 3 patients are well 7 and 6 years respectively following the operation. The third patient, a very sick youth with other foci of infection, died 14 days after operation of bronchopneumonia.

There were only 3 patients with a healed scar without activity. One of these, the second successful pericardectomy in America (operated upon by Dr. I. A. Bigger), presented a healed scar at the time of operation but returned subsequently with tuberculous peritonitis. The latter finding suggests that the pericarditis was also tuberculous in origin. The patient is well at the present time which is 11 years since the operation on the pericardium. It has been 6 years since the operation on the second patient with a healed scar and he is entirely free of symptoms. The third patient, who was operated upon less than a year ago, appears to be well. This small number of cases confirms the opinion of others that the prognosis is better with a healed scar than with an active process.

A postoperative photograph of three patients is shown in Figure 12. At the time of operation, one of these had a healed scar without activity, one had a scar resulting from a staphylococcus infection and activity was still present, and the third patient had constriction from a tuberculous process which was unhealed. These are representative of the three types of disease that we have encountered at operation. Photomicrographs of sections of the scars are shown in Figures 13, 14, and 15.

Pericardectomy was not performed, for various reasons, on 5 patients in whom the clinical impression was confirmed at autopsy. Two of these died within less than 24 hours after having been admitted to the hospital. An active tuberculous process without fluid was found in all cases and it was considered to be the chief cause of death.

An interesting unanswered question is concerned with the reason for the very slow improvement that occurs in many patients following pericardectomy. The prevailing opinion is that the constriction has resulted in atrophy of the heart muscle and that a considerable time is required for a return to the normal condition. This explanation does not suffice in all instances. At any rate, one should not despair if the improvement is slow and if a period of many months is required for the desired decline in the venous pressure and the elevation in the cardiac output.

Chronic Mediastinopericarditis

Diagnosis

Mediastinopericarditis refers to the condition in which fibrous tissue binds the heart firmly to the adjacent tissues including the

rigid structures, ribs and their cartilages, sternum, and vertebral column. The firm attachments of the heart to the neighboring structures frequently results in precordial retraction with each contraction of the heart. This is considered by most observers to be an added burden on the heart, and it may be a factor in the cardiac enlargement and heart failure which occurs not infrequently in these patients. This point is difficult to determine because mediastinopericarditis is usually associated with other abnormalities of the heart.

The type of mediastinopericarditis which may be of interest from a surgical viewpoint usually occurs as a late result of rheumatic fever. Valvular disease of the heart is usually present, and this renders difficult the recognition of the condition in patients and the understanding of the mechanical difficulties of the heart. Burwell (17) states that it is difficult or impossible to separate many of the effects of endocardial and myocardial disease on the one hand from those of pericardial disease on the other. It has been noted frequently that the largest hearts are likely to be found in association with mediastinopericarditis. Since these patients usually have valvular disease as well, it is difficult to assess the rôle played by the two disturbances. This point has been studied by Laws and Levine. They found that the average heart weight of patients with mediastinopericarditis and valvular disease is greater than that of patients with valvular disease only. The average weight of the hearts in the group with mediastinopericarditis and valvular disease was 654 grams while the average of those with valvular disease without pericarditis was 534 grams. This is interpreted as meaning that external adhesions increase the work of the heart.

Even though there are many signs which are said to be of aid, the diagnosis of mediastinopericarditis is a difficult one to make with certainty. The presence of a very large heart with multiple valve lesions should lead one to suspect it. The retraction of the precordial ribs with each systole of the heart is an important point on physical examination. Holt states that systolic retractions of the interspaces are common and without significance, being present when a large heart without adhesions is in direct contact with the chest wall. On the other hand, systolic retractions of the sternum and costal cartilages or of the eleventh and twelfth spaces in the posterior axillary usually indicate adhesions. Fluoroscopic examination may reveal a systolic tug on the diaphragm. Other signs of less value include the diastolic collapse of veins and the diastolic rebound of the chest wall. Fixation of the heart as determined by fluoroscopic and electrocardiographic studies may be of aid in diagnosis. Dieuaide (27) found when the heart is anchored by extensive adhesions that its fixation may be demonstrated by failure of the electrical axis to change to a normal degree with change of position of the patient.

Burwell (17) states that most patients with this type of pericardial disease run a course not very different from that of other patients with chronic rheumatic disease of comparable degree. In patients with mediastinopericarditis without constrictive pericarditis, the main impediment to the heart's action is with systole, not with diastole, and back-pressure with venous stasis is not a prominent finding unless myocardial failure occurs. It should be realized, however, that both constrictive pericarditis and mediastinopericarditis may be present in the same patient.

TREATMENT

There is a difference of opinion concerning the value of the several operative procedures in the treatment of mediastinopericarditis. Hosler and Williams agree with Beck in stating:

> "In view of our experiments and analysis of autopsy material, we believe that pericardial adhesions in general, and to the diaphragm in particular, do not play a significant role in the production of cardiac hypertrophy. The beneficial results of and the indications for the Brauer operation have probably been over-emphasized in the past."

Bigger (9), on the other hand, is of the opinion that some of these patients are benefited by surgical therapy. He stated:

> "The Brauer operation for mobilization of the precordial portion of the chest wall is not indicated often, and some surgeons, notably Beck, have stated that it has no place in cardiac surgery. This hardly seems correct, however, for while adhesions between

the epicardium and pericardium alone do no particular damage, such adhesions when associated with fixation of the pericardium to the chest wall or diaphragm do add a great burden to the heart, producing hypertrophy and dilatation, and eventually failure. Admittedly, such cases are rare, but when correctly diagnosed they may be relieved by this simple procedure."

Even though our experience with this disorder has been quite limited, we are inclined to agree with the opinion of Bigger. A small number of encouraging reports of benefit following the Brauer procedure have appeared in the literature. However, the assertion of Beck (2), that the extra bed rest and attention afforded these patients before and after operation did them more good than the operation itself, has not been disproved.

Even though there is some doubt as to the value of surgical procedures in the treatment of mediastinopericarditis, they will be considered briefly. If fluoroscopic examination shows evidence of dense adhesions between the diaphragm and the pericardium, the left side of the diaphragm may be paralyzed by crushing the phrenic nerve with the hope that an operation of greater magnitude will not be necessary. A temporary paralysis is preferable to a permanent one and it may be made permanent at a subsequent date if improvement follows the initial procedure.

Mobilization of the precordial area is obtained by removing cartilages, ribs, and part of the sternum. Local anesthesia usually suffices for this operation. A satisfactory incision is that which is begun over the third costal cartilage, it is directed downward and medially to the midportion of the sternum, and it ends over the seventh costal cartilage in the midclavicular line. The skin-muscle flap is dissected laterally in order to expose the bony structures. The fourth, fifth, and sixth costal cartilages are the minimum that should be removed, and it is sometimes advisable to include the third and seventh cartilages. A segment of rib, several centimeters in length, should be removed with each cartilage. In order to prevent regeneration, perichondrium and periosteum should be removed with the cartilages and ribs. It is usually advisable to remove a portion of the sternum. Bigger (36) advises the removal of the intercostal bundles corresponding to the ribs and cartilages. If it is suspected that constrictive as well as mediastinopericarditis is present, a small portion of the pericardium is exposed and an incision is made into it. If the pericardium is normal, the incision is closed. The skin-muscle flap is replaced and is closed without drainage with interrupted sutures.

SUMMARY

Twenty-eight examples of constrictive pericarditis are reported. The alterations of the circulation resulting from this condition are described. The etiology, diagnosis, course, and treatment of the 28 patients are made the basis of a consideration of these aspects of this variety of pericardial disease. For the sake of completeness there is added a brief discussion of mediastinopericarditis.

REFERENCES

1. Beck, Carl. J. Am. M. Ass., 1901, 37: 1585.
2. Beck, C. S. Am. Heart J., 1937, 14: 515.
3. Beck, C. S., and Cox, W. V. Arch. Surg., 1930, 21: 1023.
4. Beck, C. S., and Cushing, E. H. J. Am. M. Ass., 1934, 102: 1543.
5. Beck, C. S., and Griswold, R. A. Arch. Surg., 1930, 21: 1064.
6. Beck, C. S., and Isaac, Leabelle. J. Thoracic Surg., 1931, 1: 124.
7. Beck, C. S., and Mautz, F. R. Ann. Surg., 1937, 106: 525.
8. Bigger, I. A. Arch. Surg., 1932, 24: 574.
9. Idem. J. South Carolina M. Ass., 1938, 34: 147.
10. Blalock, Alfred. Arch. Surg., 1933, 26: 516.
11. Idem. J. Am. M. Ass., 1940, 114: 97.
12. Blalock, A., and Burwell, C. S. J. Lab. & Clin. Med., 1935, 21: 296.
13. Blalock, A., and Levy, S. F. J. Thoracic Surg., 1937, 7: 132.
14. Borchardt, M. J. Internat. de Chir., 1939, 4: 49.
15. Brauer, L. Arch. f. klin. Chir., 1903, 71: 258.
16. Burwell, C. S. Diseases of the Pericardium. Pp. 2580. Modern Medical Therapy in General Practice. Baltimore: Williams & Wilkins, 1940.
17. Idem. Diseases of the Pericardium. Oxford Medicine, Vol. 2, chapter 9. New York: Oxford University Press, 1940.
18. Burwell, C. S., and Blalock, A. J. Am. M. Ass., 1938, 110: 265.
19. Burwell, C. S., and Flickinger, D. Arch. Int. Med., 1935, 56: 250.
20. Burwell, C. S., Neighbors, DeW., and Regen, E. M. J. Clin. Invest., 1927, 5: 125.
21. Burwell, C. S., and Strayhorn W. D. Arch. Surg., 1932, 24: 106.
22. Chevers, Norman. Guy's Hosp. Rep., 1842, 7: 387.
23. Churchill, E. D. Arch. Surg., 1929, 19: 1457.
24. Idem. Ann. Surg., 1936, 104: 516.
25. Cohnheim, J. Lectures on general pathology. Vol. 1, New Sydenham Society, 1889

26. DELORME. Bull. et mém. Soc. de chir. de Par., 1898, 24: 918.
27. DIEUAIDE, F. R. Arch. Int. Med., 1925, 35: 362.
28. EYSTER, J. A. E. The Clinical Aspects of Venous Pressure. New York: The Macmillan Co., 1929.
29. GRISWOLD, R. A. J. Am. M. Ass., 1936, 106: 1054.
30. HARRINGTON, S. W., and BARNES, A. R. The South. Surgeon, 1940, 9: 459.
31. HARRISON, T. R., and LEONARD, B. W. J. Clin. Invest., 1926, 3: 1.
32. HARVEY, A. M., and WHITEHILL, M. R. Medicine, 1937, 16: 45.
33. HEUER, G. J., and STEWART, H. J. Surg., Gynec. & Obst., 1939, 68: 979.
34. HOLT, EVELYN. Am. J. M. Sc., 1929, 178: 615.
35. HOPE, J. Diseases of the Heart and Great Vessels. Pp. 191. London: John Churchill, 1839.
36. HORSLEY, J. S., and BIGGER, I. A. Operative Surgery. Vol. 2, chap. 42. St. Louis: C. V. Mosby Co., 1937.
37. HOSLER, R. M., and WILLIAMS, J. E. J. Thoracic Surg., 1936, 5: 629.
38. KEEFER, C. S. Ann. Int. Med., 1937, 10: 1085.
39. KUSSMAUL, A. Berl. klin. Wchnschr., 1873, 10: 433.
40. LAWS, C. L., and LEVINE, S. A. Am. J. M. Sc., 1933, 186: 833.
41. NOTH, P. H., and BARNES, A. R. Arch. Int. Med., 1940, 65: 291.
42. PICK, F. Ztschr. f. klin. Med., 1896, 29: 385.
43. REHN, L. Med. Klin., 1920, 16: 999.
44. ROBBINS, B. H., and BAXTER, J. H., Jr. J. Pharmacol. & Exper. Therap., 1937, 61: 162.
45. ROBBINS, B. H., BAXTER, J. H., Jr., and FITZHUGH, O. G. Ann. Surg., 1939, 110: 84.
46. ROESLER, HUGO. Ann. Med. Hist., 1936, 8: 353.
47. ROTHSTEIN, J. L. Arch Pediat., 1934, 51: 219; 288.
48. SCHMIEDEN, VICTOR. Surg., Gynec. & Obst., 1926, 43: 89.
49. SCHMIEDEN, V., and FISCHER, H. Ergebn. d. Chir. u Orthop., 1926, 19: 98.
50. SCHUR, MAX. Ergebn. d. inn. Med. u. Kinderh., 1934, 47: 548.
51. SMITH, H. L., and WILLIUS, F. A. Arch. Int. Med., 1932, 50: 171; 184; 192.
52. STEWART, H. J., HEUER, G. J., DEITRICK, J. E., CRANE, N. F., WATSON, R. F. and WHEELER, C. H. J. Clin. Invest., 1938, 17: 581.
53. TROUT, H. H. Arch. Surg., 1931, 23: 966.
54. VOLHARD and SCHMIEDEN. Klin. Wchnschr., 1923, 2: 5.
55. WEILL, E. Traité clinique des maladies du coeur chez les enfants. Paris: O. Doin, 1895.
56. WELLS, H. G. Am. J. M. Sc., 1902, 123: 241.
57. WHITE, P. D. Lancet, 1935, 2: 539; 597.
58. Idem. Heart Disease. New York: The Macmillan Co., 1937.
59. WILKS, SAMUEL. Guy's Hosp. Rep. 3d series, 1871, 16: 196.
60. YOUMANS, J. B. Ann. Clin. Med., 1926, 4: 1032.

EVALUATION OF INCAPACITY PRODUCED BY INJURIES OF THE PERIPHERAL NERVES

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TO evaluate the incapacity resulting from injury of a peripheral nerve, it is necessary, if the evaluation is made soon after the injury, to determine whether the nerve is anatomically severed or only physiologically interrupted; stated in another way, whether it is spontaneously recoverable or not. It is further necessary to determine whether it is completely or only partially anatomically severed. If not severed, but compressed, is it spontaneously recoverable?

If evaluation is made at a later date it is necessary to determine first whether spontaneous recovery has occurred as fully as might be expected and then to measure the residual disability. It then becomes necessary to determine whether operative treatment would be of benefit and to evaluate the probability of recovery. After operations upon the nerve, if seen some time later, it is necessary to determine whether the nerve is recovering and to evaluate the probability of further recovery and its expected degree. Opinion must be based upon the time and character of the operation and pathological condition found.

If, after a sufficient time following the operation, some disability remains, it is necessary to determine whether operative procedures other than on the nerve, such as muscle transplantation, tendon transplants, capsulotomy, may further improve function; and finally, if no further recovery is expected, to determine loss of function. It does not always follow that, because a nerve may not recover, loss of function is permanent. At times, the movements subserved by the injured nerve may be produced by other muscles, for example, almost complete recovery of movement about the shoulder girdle may take place in some cases of irreparable injury to the circumflex nerve.

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In the evaluations of residual incapacity there is found uncertainty, lack of uniformity, and discrepancies between the authors and boards of different countries and of the same country. At times the evaluation has been based upon analogous situations, for example, the loss of function produced in a part of an extremity by ankylosis may be used as an index of the loss of function resulting from an injury of a nerve, thus producing inability voluntarily to move the joint with which comparison is made. Although superior to pure guesses predicated upon which nerve is involved and at what level it is injured, such an approximation does not take into account sensory loss and pain. However, some authorities give quite divergent evaluations for ankylosis of a joint and inability voluntarily to move a joint because of paralysis; Remy, for instance, allows only 20 per cent for an ankylosis at the knee and 40 to 80 per cent for femoral nerve paralysis.

It is obvious that inability to move portions of extremities produces a degree of incapacity which is related to the particular occupation of the patient. A mild sensory loss in the hand would incapacitate a violinist and be nonsymptomatic in the case of a ditch digger. To arrive at any accurate tabulation of incapacity, it is necessary to combine the knowledge of neurologists with that of industrial surgeons and boards of compensation. To this the neurologist can contribute the knowledge necessary to determine the degree of injury of a nerve, its recoverability, and the final residual loss of function. Other specialists are necessary to determine the relation of such loss of function to the particular occupation of the patient.

ANATOMICAL SECTION AND COMPLETE PHYSIOLOGICAL INTERRUPTION

Although many attempts have been made to discover a sign or group of signs which would

justify at a single examination a differential diagnosis between a complete anatomical section of a nerve and one in which complete loss of function is not the result of discontinuity of the nerve but only a physiological interruption, none has been discovered.

Of course, if it is found that only some of the muscles supplied by a nerve are paralyzed or that only a part of the sensory area of the nerve is anesthetic or that the paralyzed muscles contract to stimuli with a faradic current, the nerve is not sectioned.

From a single examination at a particular time, we can determine only whether the lesion is complete or incomplete. If complete we cannot determine at that time whether the nerve is severed or whether its function is totally lost because of some other pathological condition. One cannot predict whether the nerve will spontaneously recover or require surgical treatment.

In a case of complete physiological interruption, only when a subsequent examination shows some return of function may we say that the lesion is incomplete. No other sign or group of signs suffice.

Complete physiological or anatomical interruption both are characterized by a complete paralysis of all of the muscles supplied by that nerve, a complete anesthesia of the isolated sensory supply of that nerve, and a complete reaction of degeneration of all of the muscles it supplies. In addition, many authors have held that the following changes are characteristic of complete lesions: rapid and extensive atrophy of the paralyzed muscles; absence of pain on pressure applied to the nerve trunk below the lesion; marked atony of the muscles; absence of any pain on pressure of the muscles supplied by the nerve; exaggerated excitability of the muscles to mechanical stimuli; abolition of corresponding reflexes; absence of any zone of hyperesthesia or paresthesia in the region supplied by the injured nerve; and severe vasomotor and trophic disturbances. None of the additional signs or combinations of them allows of a determination of the degree of injury in my opinion. Rapid and extensive atrophy of the paralyzed muscles may be interpreted as meaning a severe lesion only with a number of reservations. Ulnar nerve lesions as a rule show extensive atrophy whether severe or not. Atrophy is of service only when seen relatively soon after injury. Later it is concealed by replacement by other tissue, fibrosis, etc. Thus in an irrecoverable sciatic nerve lesion the amount of loss of mass of tissue may be 1 per cent, that of a recovering lesion, 17 per cent.

Absence of pain, in instances in which the trunk of the nerve is subjected to pressure below the seat of the lesion, was demonstrable in many of my severe lesions, but quite a number of recoverable lesions showed this as well. On the other hand, not a few irrecoverable lesions showed preservation of pain to such pressure. Only the ulnar, radial, and peroneal nerves are suitable for isolated pressure upon their trunks, and this only in cases in which the injury is proximal to their superficial positions. The hazard of producing pain by pressure upon adjacent structures is too great to make this sign significantly diagnostic; absence of pain on pressure of paralyzed muscles is even less significant. In fact in many cases tenderness to pressure was greater over the paralyzed than normal muscles. Hypotonia is too rapidly masked by fibrosis and other local changes to be of any value. Too little is known of the nature of trophic disturbances to enable us profitably to use them in interpreting the severity of a lesion. Ulceration is common in analgesic areas, generalized atrophy of bone occurs with disuse, hypertrichosis appears under bandages and casts; in fact, these changes can be of value only when judged in the light of other findings. As to the absence of hyperesthesia in the region supplied by a nerve, it may confidently be stated that hyperesthesia regularly appears when sensation to pin prick has returned in an area of nerve overlap in completely severed nerves.

The only positive evidence of a complete interruption of function of a nerve is complete paralysis of all of the muscles it supplies, a complete anesthesia of its isolated sensory supply, and a complete reaction of degeneration of the paralyzed muscles.

Total paralysis of all the muscles supplied by a nerve cannot alone be used as an indication of the severity of that lesion. Particularly is this true of the radial nerve, slight injuries

of which may produce total temporary paralysis.

Motion of segments about a joint does not indicate the integrity of the function of the nerve supplying the muscles ordinarily supposed to move such segments. The degree of preservation of the function of muscles is largely ascertained by an examination of the movements of segments about joints and not by the muscles themselves. The frequency with which more than one muscle may produce a similar movement of the segments about a joint emphasizes the necessity for the use of great care in the analysis of all muscle movements. This care is the more necessary in the study of peripheral nerve lesions because the muscles under consideration may receive their nerve supply from different sources.

The preservation of certain movements the loss of which is supposed to follow particular nerve lesions has been observed for many years. These movements may be caused by a number of factors. Among these may be included the anastomotic supply of muscles from adjacent nerves, movements produced by muscles other than primary movers in this action, movements occurring as the result of mechanical factors producing a change of direction of leverage by shortening and lengthening the tendons and muscles passing over several joints, and slight movement resulting from the recoil of elastic tissue following a movement in a direction opposite to the one desired. It is misinterpretation of such "supplementary movements" which lead to incorrect opinions that complete lesions are partial ones.

It is well known that shortly following a division of a mixed nerve, there is seen a certain area of its sensory supply in which all sensation is lost surrounded by an area in which stimuli by pin prick and extreme degrees of temperature are felt. This sensory dissociation and the subsequent behavior of the two zones led to the formulation by Head and his coworkers of their theory that the sensory mechanism in the peripheral nerves consists of three systems: deep sensibility, protopathic sensibility, and epicritic sensibility. The early return of protopathic sensibility (pin prick and extreme degrees of temperature) within the area of the anatomical sensory supply of a nerve is due to the overlap of function of adjacent uninjured nerves. This return of protopathic sensibility should never be used as an indication of a partial lesion or a recovering one. Only when sensation is present in the isolated sensory supply of a nerve can the lesion be considered a partial or recovering one. The area of the isolated supply to pin prick of various nerves is the area in which no overlapping occurs.

Corresponding to the area of the isolated supply of a nerve to pin prick is found analgesia to pinching, in my opinion an excellent indication of a severe nerve lesion.

Regarding the electrical changes demonstrated by faradic and galvanic stimulation, it may be said that complete reaction of degeneration is always present in a severe lesion but does not indicate an irreparable one. Of all the changes to electrical stimulation the slowness of the muscular contraction is the only constant phenomenon which can be satisfactorily employed in determining the reaction of degeneration. Polar changes are inconstant and of course it may be expected that response to faradism would be absent frequently even in partial lesions. Especially is this true of patients requiring more than 4 months to recover. Before a year had elapsed following injury reaction of degeneration did not indicate an irreparable lesion but after this period and always after 100 weeks, irreparable lesions show complete loss of any response to any form of electrical stimulation.

Partial and spontaneously irrecoverable lesions of nerves may also occur, for example from lateral notches. Whether these are spontaneously recoverable as in the case of complete section can be determined only by repeated examinations revealing evidence of regeneration.

SPONTANEOUSLY RECOVERING LESIONS

The signs of regeneration of a nerve are the manifestations of recovery of function. Chief among these are a return of sensibility in the isolated supply of a nerve, disappearance of reaction of degeneration, and return of motion.

These manifestations differ in appearance and rate of return as to the pathology of the nerve and as to whether recovery is spontaneous or is consequent to surgical intervention. They are dependent upon the condition of the neuraxones. If descending degeneration has been slight or absent and the nerve recovers spontaneously and rapidly one type of course is followed; if resection and suture has been performed another type is observed. If little or no degeneration has followed but a complete physiological interruption has existed for a long time, because perhaps of a constricting band, surgical relief of this morbidity is followed by a regeneration similar in character to that observed in lesions rapidly recovering spontaneously. If descending degeneration is severe or complete and conditions are such that the lesion recovers with no surgical interference, the course of recovery will be very similar to that observed following suture.

Rapidly and spontaneously recovering lesions showed two characteristics: First, in agreement with others (Sherren), I have found that such lesions do not show the dissociation of sensation previously referred to. Here little or no sensibility to pin prick returns before tactile sensation. Both forms of sensation are absent and return together. This in my opinion is due to the fact that the function of overlapping nerves is inaugurated only in the presence of a complete interruption whether it be physiological or anatomical. From 200 peripheral nerve injuries which were incomplete and recovered soon after injury, only 3 were found in which sensibility to pin prick was present and tactile sensibility absent. The return of sensibility to both pin prick and touch followed no definite rule as to its location but was in every instance patchy in character. Second, the return of function did not adhere to any definite rate of progression either as to sensation or motion and often all the muscles innervated by a nerve regained their function suddenly irrespective of their distance from the lesion.

Many cases of complete physiological interruption of a nerve showed the first sign of regeneration at such a time as one would expect it to occur were the nerve divided at the time of injury and sutured. From this time onward the regeneration progressed exactly as would a sutured nerve. It is reasonable to assume that in this type of severe lesion complete descending degeneration had occurred and conditions permitted the regeneration of the axones. Evidence of regeneration first appeared in from the eighth to ninth month, and it was noticeable that a considerable number of men wounded at about the same time, all began to improve together.

In my opinion this is additional evidence that very great conservatism should be exercised in making a decision for resection and suture in severe lesions of peripheral nerves not recovering within even 7 to 8 months.

The order in which the signs of regeneration appear have been given by Mme. Benisty as follows: (1) Sensory regeneration, consisting of pain when the skin is pinched, pain when the nerve is pressed below the lesion, formication on pressure of the nerve and spontaneous aching in certain muscles; (2) arrest of atrophy and return of tonicity; (3) in some cases return of faradic contractility; (4) disappearance of objective sensory disturbances; and (5) voluntary movements.

As critical an examination of the clinical signs of nerve regeneration is necessary as was seen to be the case with the signs of complete physiological interruption of a nerve. In my experience return of pain upon pinching of the skin was many times the first sign of nerve regeneration, but very frequently was not. Only that return of pain to pinching, which is found in such areas of skin as are outside the influence of nerve overlap, can be used as an indication of the recovery of a nerve. Frequently spontaneous aching and more frequently a sensation of a "different feeling" in an extremity preceded other signs of nerve regeneration. Pain upon pressure of the nerve trunk distal to the lesion was found unreliable.

Tinel's sign, or peripheral formication upon pressure or light percussion of the nerve trunk distal to the lesion, was found to be practically valueless. When a nerve is superficial and pressure may be exerted upon it and it alone, this sign might have some value. Unfortunately, only few of the peripheral nerves have a superficial course and this only for a short distance. Elsewhere other structures may be

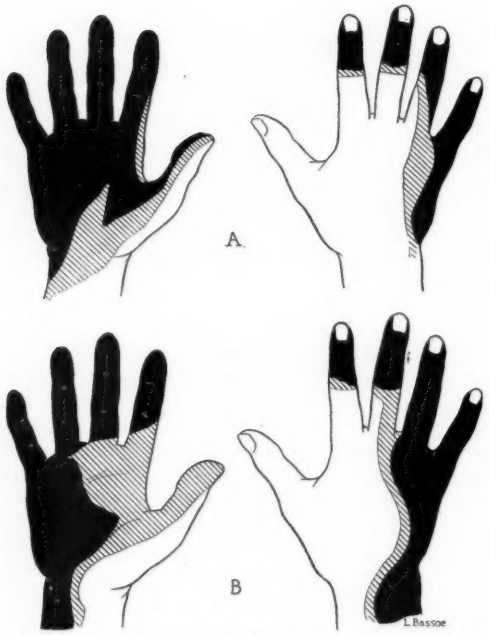

Fig. 1. Sensory loss in complete lesions of the ulnar and median nerves. Black, loss of pain, touch and temperature. Shading, loss of touch.

included in the pressure. When the sign is elicited by light percussion, the concentric waves of motion transmitted from the percussed spot may stimulate the nerve at a considerable distance.

In any event, of 50 cases of recovering lesions, 7 had complete absence of Tinel's sign and 8 had formication for only a short distance from the site of injury. Of 50 irreparable lesions, a complete Tinel's sign was obtained in more than 50 per cent of cases and only in 7 cases was it completely absent.

Arrest of atrophy and return of tonicity were not profitably employed in those nerves recovering some months after injury for reasons already stated.

The electrical phenomena of regeneration of nerves were observed only in those patients recovering more than 5 months after injury. They were not sufficiently critically studied to permit us to use the results obtained in judging the reliability of current views. It may be well to state, however, that in the partial lesions which showed beginning recovery before the eighth month following injury, a response to faradism at times returned before motion. At times motion was present and faradic response absent. Those showing beginning regeneration following resection and suture, performed not less than 6 months following injury, never showed any return of response to faradism before the return of motion. The same is true of the nerves showing beginning spontaneous regeneration only 8 months or more after the injury.

The early return of sensibility to pain and extremes of temperature in severe lesions of the peripheral nerves and which is attributable to overlap follows a pattern so characteristic that it may be recognized at sight. It never returns in the distal phalanges of the little finger in ulnar nerve lesions, never in the distal phalanges of the index and middle fingers in median nerve lesions, and so on. It always occurs along the borders of an uninjured nerve and may well be described as a shrinkage (Fig. 1).

Contrasted with this, the pattern in recovering nerves is strikingly different. Although shrinkage occurs, other changes are always present (Fig. 2).

The characteristic features of the sensory loss of regenerating nerves may be enumerated as follows: return of sensibility to pain, touch, or temperature sense in that area of the sensory distribution of a nerve which is supplied by it alone, i.e., the isolated sensory area of the nerve; return of sensibility to pain, touch, or temperature sense in patches some distance from the area supplied by an adjacent uninjured nerve; return of sensibility to pain, touch, or temperature sense in deep indentations; diminution of degree of loss of sensation of pain, touch, or temperature sense in the isolated sensory supply of a nerve; return of sensation of pain, touch, or temperature in the border between the sensory supply of two nerves simultaneously injured; interlacing of the border of sensory loss of one type of sensation with that of another.

In the interpretation of the significance of return of motion relative to regeneration, proper recognition must be made of supplementary motility. So great does this influence

the movement of some segments that I have never been able definitely to state that such movements as I have observed return following resection and suture of the ulnar nerve, were due unquestionably to nerve regeneration, with the exception of a distinct contraction of the flexor carpi ulnaris. These movements which cannot be supplemented in the various nerve lesions are:

In musculospiral lesions, extension of the proximal phalanx of the thumb and abduction of the thumb in the plane of the palm, and extension (not alone tension) of the proximal phalanges of the fingers. In ulnar nerve lesions, flexion of the proximal phalanges of the ring and little fingers with the distal phalanges extended, and lateral movements of the extended middle finger. In median nerve lesions, flexion of the distal phalanx of the index finger and of the thumb. In combined lesions of the ulnar and median nerves, all movements of the hand except flexion at the wrist and hollowing of the hand. In external popliteal lesions, eversion of the foot.

Some signs of complete motor recovery are of value: In the musculospiral nerve, placing the little finger on the seam of the trousers with the fingers well extended and with the palm turned to the front, is a sign suggested by Pitres. In ulnar nerve lesions, he suggests that the palm be placed flat upon a table with the fingers apart, then the middle finger should be moved inward and outward and finally the table scratched with the nail of the little finger without moving the wrist. In median nerve lesions Claude suggests the clenching of the fist with all the fingers well flexed into the palm and with the distal phalanx of the thumb firmly pressed upon the dorsal aspect of the second phalanx of the middle finger.

It has been observed by Mme. Ath. Benisty that following individual nerve lesions, certain muscles recover motility in a definite order. In a general way, in our experience, the spontaneous recovery showed this same characteristic: In musculospiral lesions the extensors of the wrist were the first to recover, followed by the extensors of the fingers, then the abductor and extensor of the thumb. In ulnar nerve lesions the intrinsic muscles of the hand were the last to recover. In median nerve lesions, the muscles of the thenar eminence and the flexor of the index finger were the last to recover. In external popliteal lesions the extensors of the toes and in sciatic nerve lesions the tibialis posticus and the flexors and extensors of the toes were the last to recover.

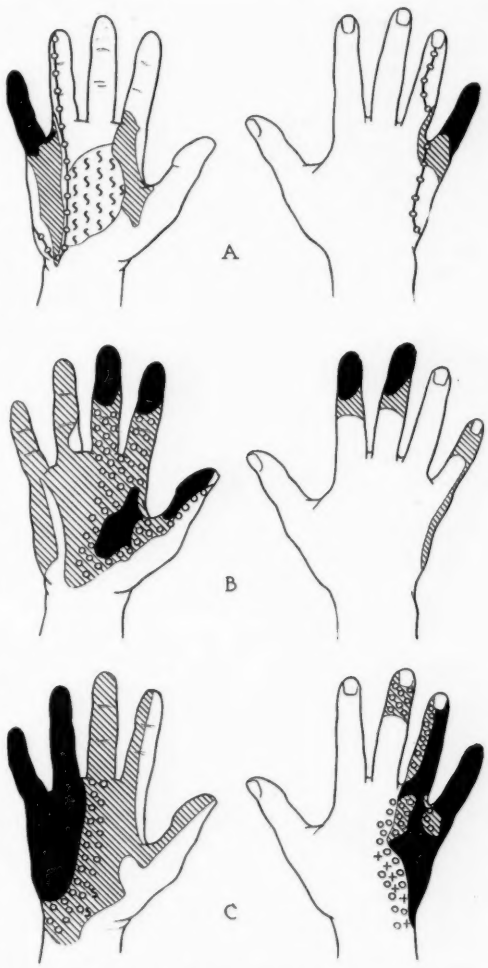

Fig. 2. Sensory loss in recovering lesions of the ulnar and median nerves. Black, loss of pain, touch and temperature. Shading, loss of touch, temperature. Letter S, hypesthesia. Letter X, analgesia.

Recovery following primary and secondary suture did not always take place according to

a definite rule. I have observed only 39 such cases and among them the first return of function occurred in the following muscles: In musculospiral lesions following primary sutures, the extensors of the wrist, the extensor communis digitorum and abductor pollicis, followed by the extensor longus pollicis; following secondary suture, extensors of wrist, followed by the extensors of the fingers, then the thumb. In median nerve lesions following secondary suture, the pronator radii teres, the palmaris longus, and flexor carpi radialis were the first to recover. The supplementary movement in ulnar nerve lesions was so extensive that it was not profitable to attempt to determine what muscle regained its function first. In combined lesions of the ulnar and median nerves, the flexor carpi ulnaris, flexor longus pollicis, and flexor sublimis digitorum were the first muscles to function following secondary suture. In the external popliteal nerve the peronei, tibialis anticus, extensor longus digitorum, and extensor hallucis were the muscles to recover function first, in the order named, following primary suture. Following secondary suture function in the tibialis anticus and extensor longus digitorum returned first. In the sciatic nerve, following primary suture, the peronei and the extensor longus digitorum were the first to recover function.

Following primary suture the first return of motion was observed in 6 months in musculospiral lesions. The first return of motion in external popliteal cases was observed in 7 months, and in sciatic nerve lesions, in 6½ months, following primary suture. In musculospiral lesions following secondary sutures, the first return of function occurred from 5 to 6 months after suture. In secondary sutures of the sciatic nerve the first return of function was apparent in 6½ months. In external popliteal nerves, the first return of function occurred in 8 months following suture. In a combined ulnar and median nerve lesion, muscles innervated by both nerves showed return of function in a little over 6 months following secondary suture.

When function is considered to be spontaneously recoverable it is necessary to know the expected chances of recovery.

In severe lesions, not anatomical sections, recovery occurred in 21.8 per cent of Wexburg's cases and improvement in 30.4 per cent. Foerster reported 60 per cent of 1,320 cases as recovered, 30 per cent improved, and 10 per cent unimproved. Herzog reported 9.4 per cent recovered and 72.7 per cent improved in lesions of the upper extremities, and 6 per cent recovered and 24.2 per cent improved in lesions of the lower extremities. The duration of the time of recovery may be quite long. The longer it takes for recovery to begin, the less perfect will recovery be, but recovery may begin as long as 2 years after injury and good results may be obtained. In spontaneously recovering lesions it is therefore necessary to wait at least 2 years before final adjudication should be made of irrecoverability.

For spontaneous recovery, the most favorable prognosis may be given in the following order of nerves: tibialis, median, musculocutaneous, axillary, ulnar, radial, peroneal, and sciatic. The most rapid recovery had been observed in the following order: radial, musculocutaneous, peroneal, tibial, median, ulnar, sciatic, axillary.

Following operation it is necessary to have some idea of the prognosis, depending on the particular operation, suture, neurolysis, or transplant, and the time between the injury and operation, such as primary and secondary suture or other procedure. Reports of recovery after secondary suture are so variable as to be interpreted with great difficulty because of the many factors involved and the different standards of recovery time of the reports.

Foerster reports 3 per cent failures in 370 cases; Stracker reports 25 per cent failures in 147 cases; Ranschburg, 64.3 per cent failures in 414 cases; Forrester-Brown, 50 per cent failure; Delagénière reported complete success in 85.9 per cent, partial success in 11.2, and failure in 3 per cent. Stopford described the results of secondary suture accurately and was able to examine his cases for a long time after operation. In 245 cases there were 30 failures; however, a number of cases in which only a return of protopathic sensibility had returned were included among the successes. Platt reported 150 cases of suture with recovery in 79 per cent.

The results of suture differ with individual nerves. Following suture, in a composite material of 6 German authors, failures were reported in 62 per cent of peroneal nerve suture—152 cases; 54 per cent of tibial—67 cases; 43 per cent of ulnar—228 cases; 31 per cent of median—198 cases; 30 per cent of sciatic—121 cases; 28 per cent of radial—397 cases; 21.4 per cent of axillary—18 cases; 18 per cent of musculocutaneous—33 cases.

The interval between the time of suture and injury is of some prognostic significance. Thus Stracker reported that of 21 cases in which operation was done within 3 months after injury, success resulted in 66 per cent; in 4 to 6 months—46 cases, 50 per cent; in 7 to 22 months—69 cases, 24 per cent.

Stopford believed that a delay of from 12 to 18 months appeared to have no marked effect upon the time or extent of recovery. If the interval exceeded this period the prognosis was not so good when the suture had been performed in the distal part of the limb. When in the proximal part a delay of 2 to 3 years does not seem to prejudice the chance of success.

It has seemed to many authors that primary suture results in relatively rapid and constantly favorable results, with which I agree.

Following neurolysis, successful results followed in 77.2 per cent of Foerster's 194 cases; in 37.2 per cent of 405 cases reported by Ranschburg; in 75 per cent of 80 cases reported by Platt.

Homoplastic and heteroplastic transplants have been uniformly unsuccessful. Of 216 cases analyzed by Gossett and Charrière, in only 5 were good results seen in autografts, 2 in homografts, and 5 in heterografts.

When after a sufficient time has elapsed following operation, 2½ to 3 years, to enable one to say that there will be no further recovery, other operative procedures should be contemplated, such as arthrodesis, tendon and muscle transplants, and similar procedures. Only when no more favorable results may be expected should the final evaluation of incapacity be made.

McBride suggests that the disability be measured in terms of loss of abduction, adduction, flexion, extension, and rotation. The disability then is approximately the same as in the presence of ankylosis. This suggestion has many merits and is far more sensible than the guesses arrived at by some. If in addition, the examiner bears in mind the extent of sensory loss as well as the type of occupation of patient, with the idea of determining what function required by that or allied occupations has been destroyed, he will be able to make a fairly accurate approximation of loss of function.

Kessler has properly noted that the estimate of permanent disability is often made at the time patient returns to work regardless of whether the nerve might regenerate further. Thus in one instance, a musculospinal lesion, patient was awarded a 30 per cent loss of function; later, however, complete recovery took place. As has been pointed out the beginning of recovery may be delayed in spontaneously recovering nerves as long as 2 years and in sutured nerves as long as 2½ to 3 years. For future reference and further to emphasize the difference of opinion concerning evaluation of incapacity produced by paralysis in various nerves, it may be of interest to note the evaluations from several sources.

Injury to the whole brachial plexus is awarded by Kaufman, when on the right side 75 per cent, on the left side 65 per cent; the *guide barême*, on the right, 70 per cent, on the left, 60 per cent. Injury to the upper part, or Erb's palsy, is awarded by Imbert 20 to 25 per cent; by *guide barême*, 20 per cent on the right, 10 per cent on the left; by Llewellyn, right 25 to 30 per cent, left, 20 to 25 per cent. Injury to the lower brachial plexus or Klumpke-Déjerine paralysis, is awarded by *guide barême* 30 per cent on the right, 20 per cent on the left; by Llewellyn 35 to 40 per cent on the right, 25 to 30 per cent on the left.

For injury to the suprascapular nerve, Llewellyn awards 10 per cent when external rotation is important; *guide barême* 30 per cent on the right and 20 per cent on the left. The subscapularis is awarded 10 to 15 per cent by Kessler. For injury to the circumflex nerve Llewellyn awards 25 to 30 per cent; *guide barême* 20 per cent on the right, 10 per cent on the left; Imbert 20 to 25 per cent; Remy 15 per cent on the right, 12 per cent on the left;

Thieme 60 per cent on the right, 50 per cent on the left; Kaufman 15 to 20 per cent; the Swiss authorities, 15 to 20 per cent; Mayet 20 per cent.

The long thoracic is awarded 15 per cent by Kaufman.

The musculocutaneous is awarded 30 per cent by Mayet; the *guide barême* awards 50 per cent on the right, 40 per cent on the left; Llewellyn likewise gives 50 per cent on the right and 40 per cent on the left for complete lesions and 30 per cent on the right and 20 per cent on the left for incomplete lesions; Kessler gives 60 per cent.

When the musculospinal is injured above the triceps, Mayet gives 50 per cent, below it, 40 per cent. When in the arm Remy gives 60 per cent on the right, 10 per cent on the left as does Imbert. The *guide barême* awards 60 per cent on the right, 50 per cent on the left, generally the German authorities give from 40 to 60 per cent. When the hand alone suffers from the paralysis Thieme gives 50 per cent on the right and 45 per cent on the left; Remy 50 per cent on the right and 48 per cent on the left; and Kaufman 50 per cent. Kessler gives 66 per cent on the hand.

For median nerve lesions above the innervation to the flexors of the wrist and fingers, the *guide barême* gives 50 per cent on the right and 40 per cent on the left, as does Remy; Imbert gives 45 per cent to 50 per cent; Llewellyn gives 60 per cent on the right, 50 per cent on the left; Thieme from 40 to 50 per cent. When involving only the small hand muscles, Remy gives 45 per cent on the right and 35 per cent on the left, and Imbert gives from 15 to 20 per cent. Kessler gives 40 to 70 per cent in the arm, 10 to 20 per cent in the hand. For causalgia Llewellyn gives 80 per cent on the right, 70 per cent on the left. For lesions of the ulnar nerve Kessler gives from 15 to 60 per cent below the elbow, 45 to 60 per cent above the elbow, 20 to 60 per cent at the elbow, in terms of hand disability; the *guide barême*, at the elbow 50 per cent on the right, 40 per cent on the left; Remy in the hand 12 per cent on the right, 10 per cent on the left; Thieme above the hand 55 per cent on the right, 50 per cent on the left; Mayet, 30 per cent above the elbow, 20 per cent below the elbow; Llewellyn, 50 per cent right, 40 per cent left, and at the wrist, 25 per cent on the right and 30 per cent on the left.

The sciatic nerve is awarded 60 to 70 per cent by Llewellyn; 60 per cent by Imbert; the German authorities generally, 40 per cent; but although Remy gives 40 per cent he thinks 60 per cent is not excessive.

The peroneal nerve is awarded 10 per cent by Remy; 20 per cent by the *guide barême;* 30 per cent by Llewellyn, 40 per cent by Golebiewski (quoted by Imbert).

The tibial nerve is awarded 20 per cent by Llewellyn.

The femoral nerve is awarded 50 to 60 per cent by Llewellyn; Imbert gives it 40 to 50 per cent; the German authorities generally, 66 to 85 per cent; the *guide barême*, 45 to 50 per cent.

The wide difference of opinion held to be authoritative is immediately apparent and often ludicrous. As Llewellyn has pointed out, to determine disability by the level of a lesion and not by actual paralysis present is certainly erroneous. If the level of the lesions is to be considered then the level should be related to the branch of the nerve above which it is located.

For ankylosis at the knee Imbert awards the same as for amputation, 60 to 65 per cent; Remy gives it as 20 per cent; Brouardel, 20 to 60 per cent according to occupation; Llewellyn, 25 to 45 per cent. If a brace is cumbersome, an arthrodesis can produce an ankylosis at the knee when the femoral nerve is injured and function will be equally as good as when the nerve is not injured, yet the German authorities give as high as 85 per cent for paralysis of the femoral nerve, and Remy, on the other hand, gives 20 per cent for ankylosis at the knee.

Thus, whereas much is known of methods of determining the recoverability of nerves, the probabilities of recovery after operation, residual paralysis, and anesthesia, possible supplementary movement, and methods of improving function, much remains to be learned concerning the estimation of incapacity as related to industry, arts and professions, produced by loss of function resulting from peripheral nerve lesions.

REFERENCES

A. Signs, Symptoms and Prognosis of Peripheral Nerve Lesions

1. BENISTY, MME. ATH. The Treatment and Repair of Nerve Lesions. Military Medical Manuals. University London Press, 1918.
2. Idem. The Clinical Forms of Nerve Lesions. Military Medical Manuals. University London Press, 1918.
3. DELAGÉNIÈRE, H. A. Surg., Gynec. & Obst., 1924, 39: 543.
4. FOERSTER, O. Handbuch d. Arztl. Erfahrungen im Weltkriege. Vol. 4. Leipzig: Schjerung, 1922.
5. FORRESTER-BROWN, M. F. Brit. M. J., Lond., 1920, 2: 467.
6. GOSSET, A., and CHARRIÈRE, J. J. de chir., Par., 1922, 19: 1.
7. HERZOG, A. Muenchen med. Wchnschr., 1917, 64: 128.
8. INGHAM, S. D., and ARNETT, J. H. Arch. Neurol. & Psychiat., 1920, 3: 107.
9. PLATT, H., and BRISTOW, W. R. Brit. J. Surg., 1923–4, 11: 535.
10. POLLOCK, LEWIS J. Arch. Neurol. & Psychiat., 1919, 2: 667.
11. Idem. Surg., Gynec. & Obst., 1929, 49: 160–166.
12. Idem. Arch. Neurol. & Psychiat., 1919, 2: 518.
13. POLLOCK, LEWIS J., and DAVIS, LOYAL. Peripheral Nerve Lesions. New York: Paul B. Hoeber, 1933.
14. RANSCHBURG, P. Wien. klin. Rundschau, 1919, 33: 223.
15. STOPFORD, J. S. B. Brain, 1920, 43: 1.
16. STRACKER, O. Wien. klin. Wchnschr., 1918, 31: 1026.
17. WEXBURG, ERWIN. Handbuch der Neurologie, Spezielle Neurologie. Vol. I. p. 23.
18. WOODS, A. H. Arch. Neurol. & Psychiat., 1919, 2: 532.

B. Disability Evaluation

1. BROUARDEL, P. Les blessures et les accidents du travail. Paris: J. B. Baillière et fils, 1906.
2. GOLEBIEWSKI, E. Atlas and Epitome of Diseases Caused by Accidents. Philadelphia: W. B. Saunders Co., 1900.
3. GUIDE barême des invalidités. Paris, 1919. Ministère de la Guerre, Service de Santé.
4. IMBERT, LEON. Accidents du travail. 2d ed. Paris: Masson et Cie., 1913.
5. KAUFMAN, C. Handbuch der Unfallmedizin. Stuttgart: F. Enke, 1925.
6. KESSLER, HENRY H. Accidental Injuries. Philadelphia: Lea & Febiger, 1931.
7. LLEWELLYN, L. J., and JONES, A. B. Pensions and the Principles of Their Evaluation. St. Louis: C. V. Mosby Co., 1919.
8. MAYET, LUCIEN. Barême pour l'évaluation sommaire de l' incapacité partielle et permanente resultant des accidents du travail. Paris: Poinat, 1928.
9. MCBRIDE, EARL D. Disability Evaluation; Principles of Treatment of Compensable Injuries. Philadelphia, London, Montreal: J. B. Lippincott Co., 1936.
10. REMY. L'évaluation des incapacités. Paris: Vigot, 1906.
11. THIEME, C. Handbuch der Unfallenkrankungen. Stuttgart: F. Enke, 1909.

THE DIAGNOSIS OF CHRONIC ENLARGEMENT OF THE LEG

With the Description of a New Syndrome

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THE subject of chronic enlargement of the leg is an interesting one chiefly from the standpoint of diagnosis. Scattered descriptions of all but one of the various conditions to be described have appeared in the literature, but there has been little organization of information, and I have been unable to find any writings which deal with the subject in regard to differential diagnosis. It is my aim to discuss this condition so as to present a guide in the investigation and diagnosis of a case of chronic enlargement of one lower extremity. I also wish to present the description of a hitherto unreported syndrome of leg enlargement. The treatment of the majority of these conditions is unsatisfactory and far from standardized, and, because I have little that is new in this respect to offer, treatment will not be considered in this article.

Unfortunately, cases of leg enlargement are seen most frequently in the female sex, and consequently the majority present themselves primarily for cosmetic reasons. In this age of silk stockings, short skirts, and glamour, one enlarged leg is a serious handicap from the psychological standpoint, even without taking into account the symptoms produced. The male sex undoubtedly shows a lower incidence of this disablement despite the fact that the cosmetic reason is negligible. The main reason for the preponderance of females is that the two groups showing the greatest number of cases, the pure lymphatic and the lymphaticovenous types, occur most commonly in females, the latter being so often a complication of pregnancy.

As there are numerous causes for the development of an enlarged leg, the subject will be discussed under the following headings: (1) congenital hypertrophy; (2) lymphatic stasis and obstruction, congenital and acquired; (3) developmental venous retardation; (4) mixed venous and lymphatic partial obstruction; (5) congenital arteriovenous fistulas; (6) miscellaneous.

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CONGENITAL HYPERTROPHY

This group includes only those cases of enlargement of the limb due to congenital overgrowth. All the component tissues of the leg are otherwise normal. There are other congenital causes for leg enlargement, but they will be discussed under their appropriate groups.

In the symmetrical development of the two sides of the body it is somewhat surprising that unilateral differences do not occur more frequently. Congenital anomalies are relatively frequent, but major variations in symmetry are rare. One manifestation of unilateral inequality is an enlarged leg. Many isolated case reports of congenital hypertrophy have appeared, and Wakefield and Hines have collected a series of 223 cases. However a careful perusal of these reports demonstrates that many of them are not true congenital hypertrophies, but are either congenital lymphedemas or congenital arteriovenous fistulas. As this latter syndrome has not been really understood until recently the difficulties in differentiation can be understood. While congenital hypertrophy undoubtedly occurs, it is not as common as was previously believed.

In another contribution to the subject, Bragman lists 4 different types of congenital hypertrophy: (1) unilateral total hypertrophy; (2) unilateral hypertrophy of one extremity; (3) crossed hypertrophy of one leg and the opposite arm; (4) individual organ hypertrophy. It is only group (2) pertaining to the leg which concerns us in this discussion.

For the diagnosis of a case of congenital hypertrophy it is essential that the history shows that the defect was noticed at birth or shortly afterward. Enlargements appearing in later life are not congenital hypertrophies. In unilateral total hypertrophy, and in crossed

hypertrophy, the diagnosis is obvious, but when only one leg is involved the diagnosis must be made by exclusion. To be able to make the diagnosis in such cases, the examiner must have in mind a clear conception of the other congenital causes leading to enlargement of the leg. These will be dealt with subsequently. An aid in confirming the clinical impression of congenital hypertrophy is the frequent finding of other congenital defects, such as spina bifida, club foot, cleft palate, etc. and later evidence of mental deficiency. Prolonged elevation of the leg results in no change in size.

LYMPHATIC STASIS AND OBSTRUCTION

This group may be subdivided into congenital and acquired varieties, and it gives the second largest number of cases of enlarged leg.

a. Congenital. There is some confusion in nomenclature in this group. The true congenital leg enlargement due to lymph stasis is known as Milroy's disease, but to be correctly placed in this category there must be a definite familial incidence.

Milroy emphasized the familial tendency and reported the lesion present in 7 successive generations in his series. Descriptions of other similar families have appeared, but most cases will not give a familial history, and are therefore more correctly designated as congenital lymphedema.

The basic factor underlying this condition is a developmental defect in the lymphatic system of the lower extremity resulting in a diffuse lymphangiomatosis with lymph stasis. This is localized in the area between skin and fascia. The clinical picture presented is that of an enlarged leg which is slightly paler than the other and which demonstrates a slight pitting edema more marked toward the end of the day. That edema is the factor concerned can be proved by continued elevation of the leg for 24 hours, when the limb will be observed to be diminished in size, more nearly approaching the normal extremity. There is no increase in length of the involved leg, no increase in warmth, no dilated superficial veins, and no ulceration or angiomas. The child appears perfectly normal except for an enlarged leg. The history in these cases must be that

Fig. 1. Lymphedema precox involving the left lower leg most marked about the ankle, but also extending slightly above the knee.

the defect was noticed at birth or shortly afterward. Although the case usually can be diagnosed clinically, when in doubt, biopsy of the skin and subcutaneous tissue will settle the question. The sections will show a spongelike appearance of the subcutaneous tissue with marked increase in the skin to fascia interval. Microscopically there will be seen a replacement of the majority of the subcutaneous fat by widely dilated lymph spaces. These spaces increase in amount toward the deep fascia. There is a varying amount of fibrous tissue present about the lymphatic channels, and especially marked as a thickening of the subcutaneous fibrous septa and the deep fascia. The lymph spaces are lined by a single layer of endothelium and are seldom larger than 2 millimeters in diameter. The microscopic appearance is entirely characteristic.

b. Acquired. There is a variety of lymphedema which probably should be grouped in the congenital section, but, because of its late development, will be classified as acquired. This is the spontaneous development of a leg swelling, starting usually about the ankle, which progresses upward slowly, and within a year usually involves the leg to the level of the knee or higher. This condition is most common in females and develops as a rule in the late teens. It has been well described by Allen who has given the syndrome the name of lymphedema precox. In a study of 93 cases

Fig. 2. A severe case of elephantiasis (acquired lymphedema) following upon successive attacks of cellulitis of the leg. The hyperkeratosis, skin excrescences, edema, and pigmentation are obvious. This leg on amputation weighed 67 pounds. (Courtesy of Dr. D. G. Webster.)

Fig. 3. A moderately advanced case of acquired lymphedema of the same etiology as that portrayed in Figure 2. (Courtesy of Dr. L. P. Ereaux.)

he found that females preponderated by 87 per cent. It usually involves one lower extremity and is aggravated by prolonged standing and activity. Figure 1 shows a moderately well developed case in a girl aged 19, the enlargement being noticed 1 year previously. The complaints, as in this case, are mainly cosmetic in basis, with the addition of some heaviness and tiredness after prolonged periods of time on the feet. Clinically and pathologically lymphedema precox presents the same picture as that of congenital lymphedema, with the exception of its later appearance.

In 1930 Reichert described an accessory method of diagnosis of edematous extremities by the use of soft tissue x-rays. In lower extremity edema due to generalized causes such as cardiac, nephritis, and hypoproteinemia, the enlargement is shown to be mainly confined to the subcutaneous area but present also throughout all the strata of the leg, whereas in lymphatic stasis and obstruction the enlargement is confined to the skin-fascia interval alone. In most cases a differentiation between the enlargement which is the result of congenital lymphedema and lymphedema precox against that which is the result of chronic lymphangitis can be made, when, in the latter, a marked subdermal diffuse fibrosis can be seen, manifested as a network of fibrous trabeculations in the subcutaneous interval.

A second variety of acquired lymphedema is that which results from the partial or complete obliteration of the normal lymphatic pathways, and it is to this group that the true cases of elephantiasis belong. This obliteration is the result of moderately severe or recurrent mild attacks of cellulitis and lymphangitis, the organism being predominantly the streptococcus. These attacks result in a progressive edema, fibrosis, and enlargement of the leg by involvement of the extrafascial tissues, the result of chronic inflammation and the fibrous obliteration of the lymphatics both far and near.

The appearance of the leg differs markedly from that of lymphedema precox. The size is usually greater and may be tremendous. Figures 2 and 3 show two good examples of the severe and the ultimate stages of the condition. In both, the result is due to waves of subacute cellulitis and lymphangitis progressing for more than 5 years. Both were natives of Montreal and repeated examinations for filaria were negative. The skin shows a marked thickening and loss of elasticity, and in severe cases like those pictured here, there are patches of hyperkeratosis, pigmentation, and wart-like excrescences. The enlargement may be such that the skin bulges in hard folds with deep creases between. There is always a hard pit-

Fig. 4. This patient has had varicose veins for the past 30 years, but the enlargement of the leg followed an abrasion to the left shin. (The scar of this can still be seen.) The abrasion healed very slowly, and was associated with many attacks of subacute inflammation. This case is one of mild acquired lymphedema with pre-existing varicose veins.

ting edema present which is reduced somewhat on prolonged elevation. The hardness of the tissues is characteristic. There is usually a patchy slight increase in warmth of the involved, over the uninvolved, leg because of the subacute inflammation present in various areas. Leg ulcers are seldom present. The finding of associated varicose veins should not lead the diagnosis astray, such as the case pictured in Figure 4. This man has had veins the size of those present in the photograph for the past 30 years. Eleven years ago he had an abrasion to his lower leg and ever since has had attacks or waves of redness, tenderness, and increasing swelling of his leg. Chills were present on a few occasions. This is an example of a milder degree of chronic lymphatic obstruction. The classical example of this variety of leg enlargement is that seen in filariasis in which the elephantiasis is not caused merely by the presence of the filaria in the lymphatic vessels, but mainly by the lymphangitis which results from their invasion.

Matas and Homans have given excellent descriptions of the whole process, and have shown that mere interruption of lymphatic pathways is not enough to produce lasting edema, but for the edema to be continuous an added chronic or recurrent lymphangitis must be present.

Fig. 5. The case of congenital venous retardation described in the text, showing the slight but definite enlargement of the left leg. There is a generalized patchy cyanosis best seen in the upper thigh and ankle.

The microscopic picture of a section of skin and subcutaneous tissue demonstrates the combined appearance of lymphedema and chronic inflammation. The epiderm is seen to be hypertrophic and may be thrown into irregular folds. There are varying degrees of papillary excrescences and hyperkeratinization. The corium is fibrous and from it fibrous bands extend down through the subcutaneous tissue. The blood channels are thickened and surrounded by a perivascular exudation of lymphocytes. The fibrous tissue is loosely arranged and appears edematous. In severe cases the picture may simulate a myxomatous lesion.

DEVELOPMENTAL VENOUS RETARDATION

Pure venous retardation as a cause of leg enlargement in a previously unaffected leg has not been described to date. In other cases in which increased leg size has the factor of retarded venous return, there is also partial lymphatic blockage. This combination is well known and follows on a previous deep thrombophlebitis. This combination and its diagnosis will be discussed subsequently under group 4 as the lymphaticovenous group.

First of all I wish to present a case which in my opinion demonstrates that venous retardation can be a cause in itself of leg enlargement.

A.C., Hosp. No. 111,154, female aged 21 years, presented herself because of an enlarged left leg

Fig. 6 Fig. 7

Fig. 6. Roentgenograms of the case shown in Figure 5 following injection of 70 per cent diodrast solution into the left femoral vein at the level of the groin. The retrograde filling of the entire femoral vein and tributaries is well seen. The large caliber of the vein and the smooth walls indicate the absence of any previous deep phlebitis.

Fig. 7. Injection of the right femoral vein at the level of the groin in the same case as that shown in Figures 5 and 6. Note that the diodrast solution does not descend farther than the first valve station.

which was first noticed 1 year ago. There were no associated symptoms until about 2 months before admission when she noticed that in the afternoons, after being on her feet all day, she would have a dull aching pain throughout the entire length of the leg. The leg would be slightly larger in the evenings than the mornings but there has been no return to normal size at any time since the onset.

The patient gives no familial history of enlargement of the leg. She is married but has had no pregnancies. Before her marriage she was a waitress for 2 years. There is no history of accident to the leg or phlebitis, and she has had no operations or serious illnesses.

There were no findings in the general physical examination relevant to the local condition. The left leg showed a mild generalized enlargement in comparison with the right (Fig. 5). Even in the supine position, and much more marked when erect, the left leg showed a definite cyanotic mottling which seemed to be most marked on the lateral aspect of the mid thigh and again in the region of the medial malleolus. There were no varicose or unusually dilated superficial veins present; no nevi or areas of pigmentation; no pitting edema. The skin texture and the feel of the subcutaneous fat were normal.

The arterial pulsations were normal in both feet and legs. Measurement of the extremities revealed equal length, but demonstrated the enlargement on the left side to be due to an increase in circumference of 1 inch in the thigh and ½ inch in the lower leg. Skin temperature readings by means of the derma-

therm were essentially equal on both legs. Routine urine and blood Wassermann examinations were negative. Soft tissue roentgenograms made of both legs showed the bones to be normal in contour and length, and the enlargement of the left leg to be composed chiefly of an increased thickness of the subcutaneous tissues. There was no arterial calcification or localized zones of soft tissue increase.

The femoral vein on the left side was injected with 70 per cent diodrast solution, the technique being as follows: Venipuncture was performed just below the inguinal ligament with the patient lying on the x-ray table. An assistant then compressed the femoral vein and artery against the superior ramus of the pubis. The injection was carried out without undue pressure, the 30 cubic centimeters being given in about 30 seconds. The proximal digital pressure was continued until the roentgenograms had been taken, and the whole procedure was completed in about 2 minutes. This same procedure was carried out subsequently on the normal leg. The x-ray results are shown in Figure 6 in which it will be seen that the solution descended retrogradely almost the full length of the deep veins in the involved leg and also into many of the tributaries, but there was no reversal of flow in the great saphenous vein. The injection into the femoral vein on the normal side was halted within an inch and a half from the point of injection (Fig. 7).

The conclusions in this case are based on the following points: This patient gives no history of any previous leg disorder, she has never had phlebitis, pregnancy, or any severe illness. There is no familial history, and the enlargement has gradually appeared in early adult life, a time when the equivalent state of defective vein valves in the saphenous systems usually makes its appearance. Edema as an important factor in the enlargement can be ruled out by the absence of pitting on pressure, and only the slight diminution in size on prolonged elevation. There were none of the signs of a congenital arteriovenous fistula present. The diffuse cyanosis of the leg, most marked on standing, indicates a venous congestion; and the lack of enlarged superficial veins rules out incompetency of the saphenous system, although this will probably develop later.

The x-ray studies corroborate the clinical impression by revealing the absence of, or defective valves in, the deep system of the involved leg, thus allowing complete retrograde flow. This defect is absent in the normal leg, the injection being soon halted presumably at the first valve station. This phenomenon of retrograde flow has been present on x-ray in 2 other patients who had had a previous thrombophlebitis, whereas 2 normal controls again showed the injection fluid to penetrate a short distance down the vein. The leg hypertrophy is probably the result of venous retardation and increased venous pressure acting over a long period in a dilated deep venous bed. Undoubtedly this extremity has been endowed with a set of valves congenitally weaker, and likely numerically fewer, than the normal, and the preceding years of activity while a waitress produced their complete defectiveness.

MIXED LYMPHATIC AND VENOUS PARTIAL OBSTRUCTION

As before mentioned, to this group belongs the largest number of cases. The leg enlargement is usually only a secondary complaint with the exception of the ankle edema which appears toward the end of a day of activity. It is most commonly the complications which bring these cases for treatment. The underlying pathological condition is that of a thrombophlebitis of the deep femoral system which in many cases progresses well up into the iliac veins. This is an infective phlebitis following pregnancy most frequently, but seen also in septicemia, pneumonia, typhoid, and after operation in septic cases. This thrombophlebitis is always associated with a periphlebitis, a point which can be demonstrated at a later date when some operative procedure on the veins is carried out. Instead of the usual fine areolar tissue surrounding the vein, thick fibrous bands will be found closely binding the thickened whitish vein to the surrounding tissues. This periphlebitis results in a lymphangitis due to the close anatomical association of the lymphatics with the veins, especially in the region of the external iliac vein. Subsequent recanalization of the vein in part is almost invariable, but the action of the valves has been destroyed.

A careful history taking is important in determining a previous deep thrombophlebitis. Such a case will give a history of an acute leg involvement associated with pain, blueness, tenderness, a sharp febrile course, and, especially, marked swelling of the whole leg. These patients will have been in bed for from 4 to 8 weeks and will have been treated usually by

Fig. 8. A case of mild enlargement of the right lower leg following a femoral thrombophlebitis associated with childbirth 14 years before. The common complication of ulceration of the lower leg is present.

elevation and hot fomentations. Such a history will distinguish a deep phlebitis from the superficial vein involvement; in the presence of the latter, localized pain and tenderness, mild or absent fever, and little or no ankle edema will be elicited.

The diagnosis of this variety of leg enlargement presents few difficulties. The history of a previous deep thrombophlebitis is of paramount importance. The clinical examination reveals only a slight increase in size of the involved leg over the normal one. This is always more marked toward evening after a day of activity, especially about the foot and ankle. The leg is only slightly larger than its fellow and the most marked tissue changes are in the lower half of the lower leg. The skin in this region is indurated and usually pigmented, and the subcutaneous tissues show a distinct firmness even to the point of hardness. There is no increase in warmth unless secondary infection from complications is present (Fig. 8). The arteries pulsate normally. The appearance of the lower leg will be modified by the frequent complications of eczema and ulceration which ensue. Contrary to the popular belief, it is the exception for a case of deep femoral phlebitis to develop subsequent incompetent superficial varicose veins. Careful questioning will reveal that when varicose veins are present, they were in existence before the onset of the deep phlebitis and probably were a factor in its development.

CONGENITAL ARTERIOVENOUS FISTULA

This variety of leg enlargement is one of unusual interest. It consists of a hypertrophy of all the component parts of the leg the result of abnormal arteriovenous communications of congenital origin. As would be expected, the majority of cases develop in early childhood, but there are some which do not make themselves evident until later in life probably because of the small size of the fistulas at birth. As growth and activity continues these small fistulas enlarge to the point of producing signs and symptoms. To obtain a hypertrophied leg in a case of arteriovenous fistula, the communications must be well above the knee. The level of the fistula will determine the amount

Fig. 9. a, left, Case of congenital arteriovenous fistula involving the vessels in the left thigh, and showing the generalized enlargement of the left leg. b, Infrared photograph of the same case demonstrating the dilated skin veins in the left as compared with the right leg. Operation confirmed the arteriographic findings of two arteriovenous fistulas between the femoral artery and vein in Hunter's canal.

of leg to be involved. In the past because of the numerous clinical manifestations these different degrees were not recognized as arising from a single cause. Numerous names were applied, such as phlebarteriectasis, Parkes Weber syndrome, etc., and it is to Lewis and Horton that credit should be given for a clear definition of the underlying pathology and its clinical recognition.

The diagnosis of leg enlargement due to congenital arteriovenous fistula should not present any difficulties if the condition is kept in mind. The salient features on examination are: the leg is definitely but not greatly enlarged and is elongated if the fistulas have shown evidence of their presence before closure of the epiphyseal lines.

This elongation if at all marked will give a lifting of the pelvis on that side and a compensatory scoliosis. There is always an increased prominence of the superficial veins in the involved leg which may progress to the point of showing marked varicosities, and the common varicose vein complications of pigmentation, eczema, and ulceration of the lower leg.

Figure 9 shows an early stage of the condition with the increased superficial veins well demonstrated by infrared photography. This case has been described in a previous communication (6). A constant finding is a uniform increase in warmth of the extremity being 1 to 3 degrees C. more than the normal leg. The arterial pulsation in the vessels of the feet is either diminished or absent. A clinical test which is occasionally present is that known as Bragman's sign; that is, occlusion of the artery proximal to the fistula gives a sudden decrease in the pulse rate of 10 to 15 beats per minute. This is more commonly found in the acquired arteriovenous fistulas, and the same can be said for cardiac hypertrophy and failure which follow a well developed arteriovenous communication. These findings will give a tentative diagnosis of the condition and confirmation can be obtained by two further procedures. Arteriography carried out by means of thorotrast or preferably supersaturated diodrast solution will demonstrate the fistulous connections. Examination of the oxygen content of the returning venous blood

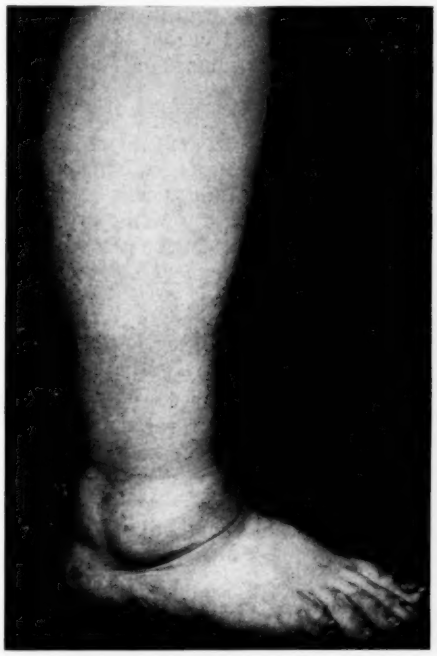

Fig. 11. The right lower leg of a patient showing plexiform neurofibromatosis showing the characteristic greater involvement about the ankle region. This case showed generalized signs of neurofibromatosis.

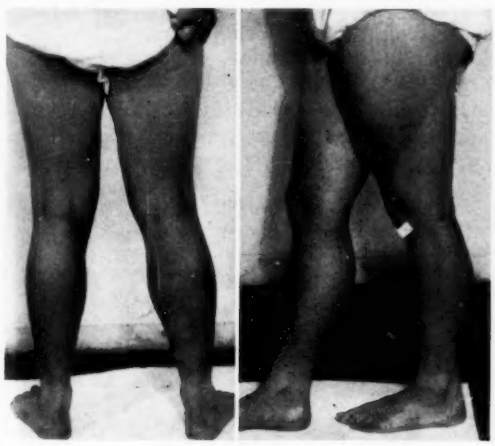

Fig. 10. A case of pseudohypertrophic muscular dystrophy involving the right calf in a girl aged 14 years. The condition did not involve any other muscle groups.

central to the fistulas will give much higher values than in normal venous blood, indicating a direct arteriovenous shunt.

MISCELLANEOUS

Two separate causes of leg enlargement will be discussed in this group: pseudohypertrophic muscular dystrophy and neurofibromatosis. The former is one variety of the myopathies and should be included in the present discussion because of the frequent commencement of the disease in the muscles of the calf. The history is usually familial and the onset is noticed between the ages of 5 and 15 years. The first symptom if the leg is involved is enlargement of the calf muscles. The condition tends to progress and, in the later stages, if the shoulder and spine muscles are involved there should be little difficulty in diagnosis. The main symptom is a weakness and difficulty in gait giving a marked limp because of increasing incompetence of the involved musculature.

Examination of an early case demonstrates symmetrical enlargement of the calf muscles. The remainder of the leg appears perfectly normal. There are none of the signs such as are present in lymphatic blockage, venous retardation, or arteriovenous fistulas. The clinical impression is that of a diffuse soft tissue mass in the calf area. Soft tissue films show the enlargement in the calf to be beneath the deep fascia. In the case shown in Figure 10, a diagnosis of subfascial lipoma was made; and, at the operation, the findings were solely those of muscle enlargement, which seemed confined almost entirely to the gastrocnemius. Biopsy showed a definite hypertrophy of the fibers and also the deposition of an abnormally large amount of fat in this muscle.

Neurofibromatosis results in leg enlargement from two main varieties, first, from numerous scattered neurofibromas, and second from the plexiform variety. The latter is the more pertinent to our discussion as it causes a diffuse enlargement extending from the foot almost to the knee, usually with folds which droop down overhanging those of the ankle in a hood-like manner. The skin is the typical *café au lait* color and, with the subcutaneous tissue, gives a peculiar putty-like feel. When neurofibromatous overgrowth involves an extremity, associated disturbances in the bones may occur. There may be a mild to massive increase in length and thickness of the long bones together with irregularity in outline which may be of such a degree as to give tumorlike projections and cystic cavities which, however, are found to consist of neurofibromatous tissue. In a recent case of plexiform neurofibromatous enlargement of the lower leg (Fig. 11), a plastic procedure was carried out, and the subcutaneous tissue was found to be extremely vascular and grossly to have the usual appearance of firm whitish jelly. The diagnosis in this group of enlarged legs is obvious because there is invariably associated with it a more or less generalized neurofibromatosis.

SUMMARY

1. This presentation is an attempt to organize the scattered information on the subject of the unilateral, chronic, enlarged leg so as to present a guide in its diagnosis. The subject of treatment has been excluded.

2. Six main groups are described, each different from the standpoint of etiology; they are congenital hypertrophy; lymphatic stasis and obstruction, both congenital and acquired; developmental venous retardation; mixed lymphatic and venous partial obstruction; congenital arteriovenous fistulas; and miscellaneous.

3. Group three, that of developmental venous retardation, is presented, as far as can be discovered, for the first time and a case history is given in detail.

REFERENCES

1. Allen, E. V. Arch. Int. Med., 1934, 54: 606.
2. Bragman, L. J. Arch. Pediat., 1935, 52: 53.
3. Homans, J., and Zollinger, R. Arch. Surg., 1929, 18: 992.
4. Horton, B. T. J. Am. M. Ass., 1932, 98: 373.
5. Lewis, D. Lancet, 1930, 2: 621, 680.
6. Luke, J. C. Canadian M. Ass. J., 1940, 42: 341.
7. Matas, R. Am. J. Trop. Dis. & Prev. Med., 1913, 1: 60.
8. Milroy, F. W. New York M. J., 1892, 56: 505.
9. Reichert, F. L. Arch. Surg., 1930, 20: 543.
10. Wakefield, E. G., and Hines, E. A. Am. J. M. Sc., 1933, 185: 493.

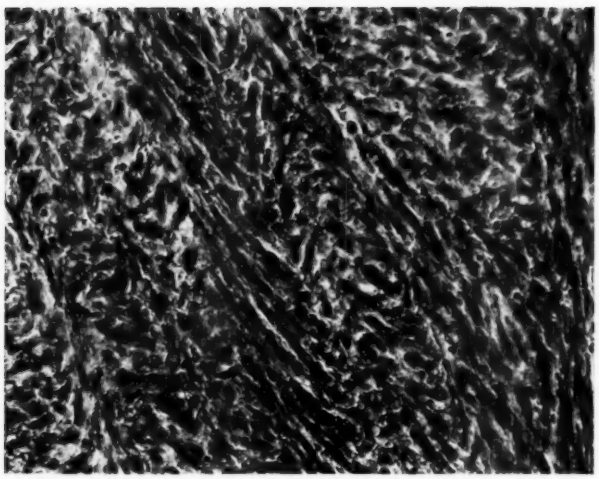

Fig. 4. Sudan stain frozen section revealing characteristic fat droplets in cell protoplasm and lying free in the interstitial tissue. ×340. (Photography by Clarence B. Mitchell.)

Theca Cell Tumors (Thecoma).—Arthur H. Curtis.

THECA CELL TUMORS (THECOMA)

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THESE tumors, first described by Moretti and Arrigoni (1927) as fibroma thecocellulare xanthomatodes ovarii, and by Loeffler and Priesel (1932 and 1934) as theca cell tumors of the ovary, are so closely related to granulosa cell tumors that many consider them a variety of the latter. Only 33 cases of theca cell tumor, aside from the one herewith described, have been recorded in the literature; yet there is every reason to believe that these growths, although uncommon, are much less rare than this figure would indicate.

Theca cell tumors are characteristically firm, fibroma-like, nodular, solid or partially cystic, unilateral growths. The tumors are usually not larger than an egg but sometimes attain the size of a small melon. The fundamental characteristic is a connective tissue derivation with a high lipoid content. These solid neoplasms arise in the ovary, develop large nodules, and encroach upon and replace the normal tissue.

On gross section, the cut surface is seen to be composed of bands and whorls of fibrous tissue which may be uniformly yellow or there may be yellow islands or streaks of yellowish color. Cysts, an inconstant feature, may have a hemorrhagic content and are commonly the result of necrosis.

Under the microscope the grossly yellow tumor is found to consist of firmly constructed bands of stroma cells varied from spindle-shape to epithelioid form, also a varying proportion of polygonal cells. The stroma cell nuclei are relatively rich in chromatin, uniform in size, and very numerous. The stroma is usually a basket weave with intercellular fibrils surrounding and intermingling with islands of more or less polygonal cells, the latter really an integral part of the growth together with the stroma, although apparently enmeshed in it. Although the spindle cells usually comprise the major portion of the tumor structure, the nests or large areas of polygonal cells with deeply staining nuclei are a frequent and characteristic finding. The latter cells commonly show some evidence of luteinization, some nests of cells, when viewed under high power, simulating the luteinization encountered in the wall of a lutein cyst (Fig. 5) or even suggestive of the structure of a corpus luteum.

One must not be confused over the relationship of the spindle-shaped stroma cells, the polygonal cells, and the luteinized cells. They are part and parcel of the same structure, varied in degree according to the cellular richness of the growth and the degree of luteinization; the richer the cellular growth, the greater the differentiation in an epithelial direction. The identity in derivation of these various types of cells is evidenced by the Mallory stain, and demonstrated still better by a modified Masson stain. The nature of thecoma tumors has been concisely stated by Loeffler and Priesel: "It is immaterial whether the structure is more a spindle-celled fascicular one or the cells are more of an epithelioid form. The ground type is always a cell of connective tissue derivation with high lipoid content."

Despite the firm fibrotic character of most of these growths, numerous thin-walled blood vessels are usually found. Poorly staining hyaline areas in the stroma are so commonly present that they are an almost characteristic feature. Even more noteworthy are minute, scattered, unstained spaces both in and between the cells; in frozen sections prepared with sudan stain these are found to contain fat droplets, not only present in the cell protoplasm throughout, but also lying free in the interstitial tissue.

Our case is that of a nulliparous married woman who continued to menstruate regularly and rather freely despite her age of 52 years. Aside from a fixed very hard tender swelling in the region of the left ovary the only other noteworthy item in the history

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Fig. 1. Theca cell tumor of ovary. This was a smoothly nodular, very firm tumor. The cut surface revealed a markedly yellow, smooth, fibrotic tumor, surrounded by a crescent of unusually firm ovarian tissue. × 1.23.

was a recently developed adenoma of the thyroid gland.

At operation, November 29, 1938, there were encountered a marked brawny fibrosis of the cellular tissues of the pelvis, scattered adhesions, multiple small fibroids of the uterus, and a somewhat adherent, tumorous, smoothly nodular left ovary.

The left ovary measured 4 by 3 by 3 centimeters (Fig. 1). The surface was smoothly nodular, board-like in firmness. The cut surface revealed $^{5}/_{6}$ of the substance converted into a markedly yellow, smooth, fibrotic tumor, surrounding which was a crescent of unusually firm ovarian tissue. There were no cystic cavities and no other gross evidence of degeneration or necrosis.

Histology. The tumor is mostly encapsulated, but in one broad area the fibrous stroma of the ovary passes over into the tumor tissue without a discernible zone of demarcation. The new-growth is composed of firm strands of spindle cells with a tendency to create a basket weave, with inter-

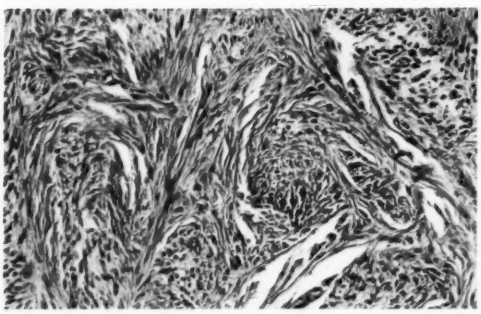

Fig. 2. The tumor is composed of firm strands of spindle cells with a tendency to create a basket weave with intercellular fibrils surrounding and intermingling with clusters of cells having numerous deeply staining nuclei. ×160.

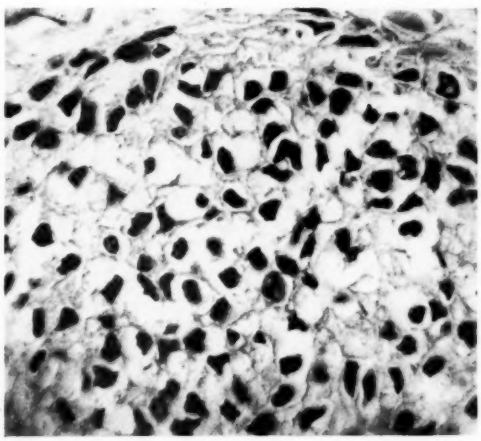

Fig. 3. High power photomicrograph of cluster of characteristic polygonal cells with epithelioid tendency. ×335.

cellular fibrils surrounding and intermingling with clusters and larger areas of cells having numerous deeply staining nuclei. These cells are polygonal in many areas, in some the cell boundaries are less clearly defined, and in still other regions the clusters of cells show definite evidence of luteinization. Much new-growth of intercellular fibrillar material is strongly in evidence.

In many areas the tumor consists largely of spindle-celled fasciculi, in others the stroma is more epithelioid, with clearly definable transition from the spindle-cell to the epithelioid-cell type. The identity of the elements comprising the tumor is well shown by a modified Masson stain. In sections stained with hematoxylin and eosin are many minute unstained spaces, also displaced individual cell nuclei, leaving clear spaces (fat). Sudan stain of frozen sections reveals innumerable fat droplets in cell protoplasm and lying free in interstitial tissue (Fig. 4, frontispiece).

Chemical analysis of a theca cell tumor shows a moderate amount of lipoid material and a high content of cholestrin. Extraction yields a large amount of estrogen per gram of tumor tissue.

Most of these tumors are in women beyond the menopause. Atypical bleeding in these postmenopausal patients is a characteristic clinical feature. The uterus is enlarged and the endometrium hyperplastic as a result of the elaboration of estrogen by the embryonic tumor cells.

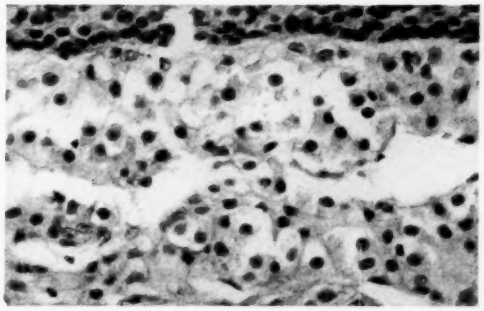

Fig. 5. Section through wall of a large lutein cyst of the ovary with typical luteinization of the theca interna, from a patient with hydatid mole. At the top are seen the granulosa cells which line the cyst cavity. Compare with thecoma cells of Figure 3. ×310.

Malignancy may occur but is rare in this kind of tumor in all except the richly cellular type of growth. Unilateral ovariectomy is the treatment of choice.

REFERENCES

1. Geist, Samuel H., and Gaines, Joseph A. Am. J. Obst. & Gynec., 1938, 35: 39.
2. Loeffler, E., and Priesel, A. Beitr z. path. Anat., 1932, 90: 199; Wien. med. Wchnschr., 1934, 84: 400.
3. Moretti, Giulio, and Arrigoni, Prospero. Folia gynaec. (Genova), 1927, 24: 33.

Fig. 5. An incision has been made around the esophageal hiatus and the esophagus is being freed by blunt dissection up into the mediastinum.

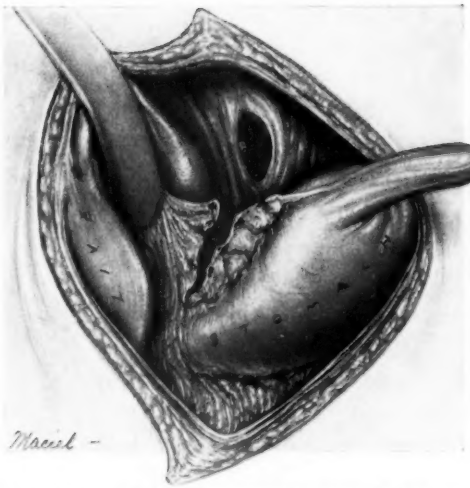

Fig. 7. The lesser curvature has been divided so that the stomach may be mobilized.

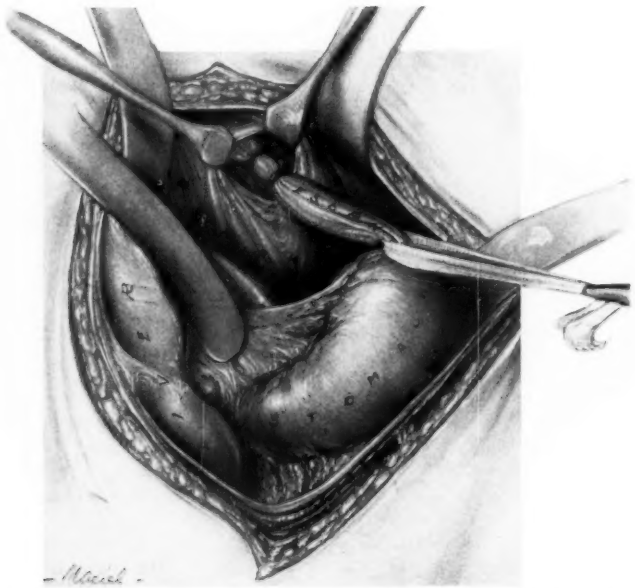

Fig. 6. The esophagus has been freed high into the mediastinum and has been doubly ligated with silk.

AN OPERATION FOR THE CURE OF CONGENITAL ATRESIA OF THE ESOPHAGUS

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THE ideal operation for the cure of congenital atresia of the esophagus is one in which a direct attack is made on that portion of the gullet which is deformed and the continuity of the gastrointestinal tract is re-established by an end-to-end anastomosis of the proximal and distal segments of the esophagus. This ideal has been striven for at the Children's Hospital in Boston as reported by Lanman by means of an extrapleural approach through the posterior mediastinum with the result that all of the patients operated upon succumbed, although 1 patient in a total of 5 survived for 8 days and 1 for 9. The remainder were apparently unable to withstand the operative procedure. Probably the next operation in the order of desirability would be one in which the deformed portion of the esophagus is attacked directly either transpleurally or extrapleurally, but in which no attempt is made to re-establish the continuity of the esophagus. The ends of one or both segments of the esophagus are brought out and later continuity is established by reconstructive operations. The Boston Children's Hospital group reported 12 such operations with 100 per cent mortality, and Richter in 1913 reported 2 cases in which, by a transpleural approach, the fistula between esophagus and trachea was ligated, both of which terminated fatally. It would seem to us from these reports, which are the only ones we have found, that the mortality resulting from operations in which the deformed portion of the esophagus is attacked directly is too high to justify their use. We have had no experience with this type of operation and our opinion is based entirely on what has been learned from the work of other surgeons.

In view of the high mortality which results from a direct attack on the deformed portion of the esophagus, several different operations have been advocated. These operations do not disturb the communication between trachea and esophagus, but attempt to form a new channel whereby food can pass from the proximal segment of the esophagus to the stomach around the fistula mentioned. In the light of our present knowledge it would seem that this type of operation is the most likely to succeed, since the operation can be done in several stages, no one of which is too shocking and, since if leakage occurs, no fatal effects follow. Such an operation should fulfill the following requisites: (a) The blind proximal segment of the esophagus must be drained to the outside. (This is essential for it is the overflow of secretions from this pouch which is responsible for death from pneumonia in nearly all of these patients.) (b) The passage of stomach contents through the lower segment of the esophagus which communicates with the trachea must be prevented, *but* a long blind pouch of esophagus communicating with the trachea must not remain; otherwise, the secretions from the pouch of esophagus will spill over into the bronchial tree and cause fatal pneumonia. (c) A satisfactorily functioning gastrostomy must be made through which food can be given, but only after the passageway between the distal segment of the esophagus and trachea has been closed. (d) The stomas of the upper segment of the esophagus and the stomach must be as close together as possible to facilitate the later joining of those two stomas by a skin tube.

It does not appear to us that the operations which have been advocated fulfill all of these requirements, nor does the procedure which is advocated in this paper do so completely, but it appears to come closest to such fulfillment. The objections to the operation proposed by Gage are that it leaves a long pouch of the distal esophageal segment still in communication with the trachea and that it leaves the

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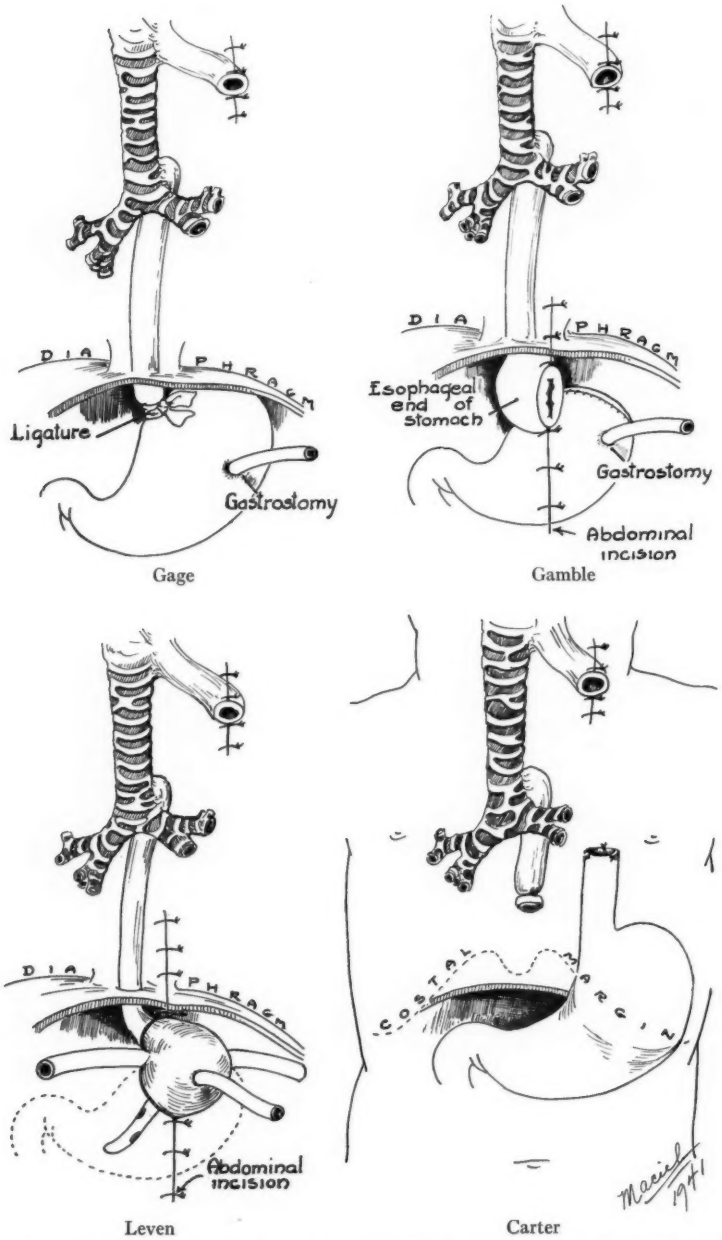

Fig. 1. Diagrammatic drawings of the operations by which the tracheoesophageal fistula is undisturbed but the flow of stomach content through it is prevented, and in which it is planned to create a new channel for the passage of food from the mouth to the stomach.

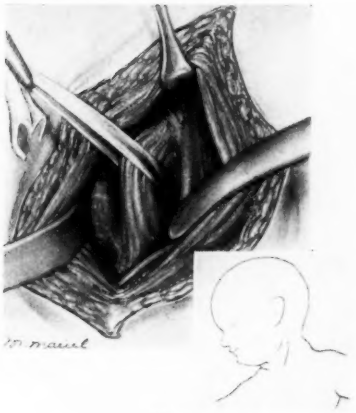

Fig. 2

Fig. 3

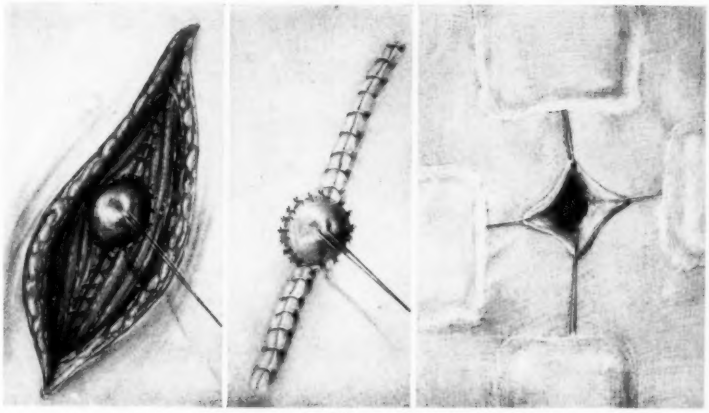

Fig. 4

Fig. 2. An incision has been made along the anterior border of the left sternomastoid muscle. The proximal segment of the esophagus has been freed and a tape has been passed around it.

Fig. 3. The proximal segment of the esophagus has been freed by blunt dissection and pulled up out of the thorax.

Fig. 4. The edges of the muscles and of the skin have been sewed to the esophageal stump, the protruding end of which has been surrounded with strips of collodion gauze. The esophageal stump has been opened and held with 4 traction sutures which have been imbedded in the collodion.

stoma of the proximal end of the esophagus far distant from that of the stomach (Fig. 1). Gamble's ingenious operation (Fig. 1) provides for drainage to the outside of the lower segment of the gullet which is in communication with the trachea but sacrifices one-fourth of the stomach to do so and the gastrostomy is separated from the esophageal stoma by a considerable distance. Also, the position of the stoma of the distal esophageal segment would interfere considerably with the anastomosis of a skin tube with the gastrostomy. A further objection is said by Lanman to be the respiratory difficulty resulting from the direct communication of the fistula into the trachea with the outside by way of the exteriorized lower esophageal segment. Leven's operation (Fig. 1) is open to much the

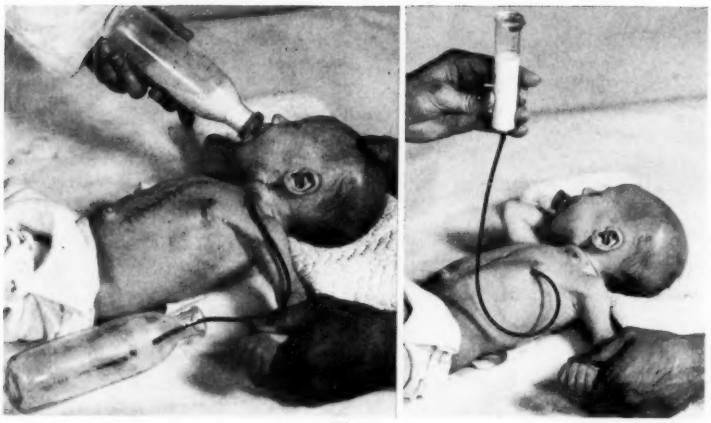

Fig. 9

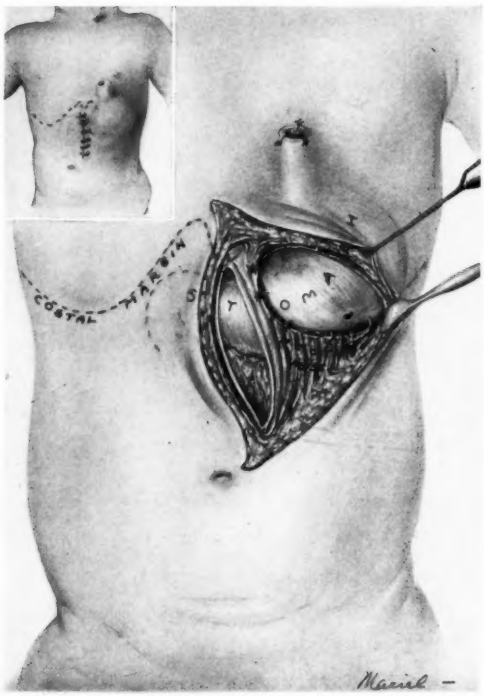

Fig. 8

Fig. 8. The skin along the left side of the rectus incision has been undermined to above the costal border and retracted. The stomach with the attached portion of the esophagus has been brought out of the abdomen through an incision just below the costal margin and placed in a subcutaneous tunnel terminating at the level of the nipple.

Fig. 9, left. The feeding taken by mouth is collected in a bottle from upper esophageal stoma, and, right, is then introduced through lower esophageal stoma into stomach.

same objections as Gamble's and, in addition, the danger of pressure slough by the tube which is placed beneath the exteriorized esophagus and cardiac end of the stomach would seem very real.

In our experience, 6 cases, the most immediate danger to which infants with congenital

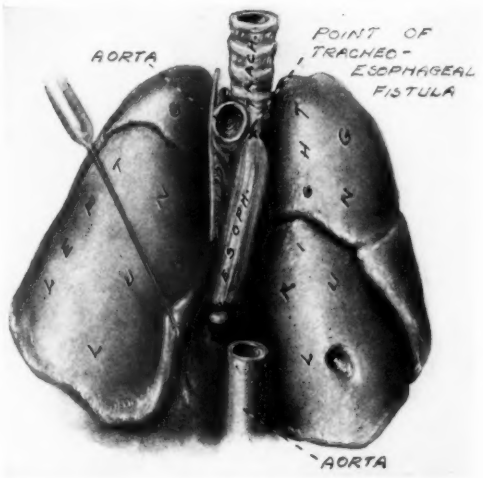

Fig. 10

Fig. 10. A drawing made of the autopsy specimen. The blind esophageal pouch was intact and no sign of leakage from it could be discovered. Both lungs showed extensive pneumonia with a bronchial fistula on the right at site of perforation of a subpleural lung abscess.

atresia of the esophagus are exposed is pneumonia caused by aspiration of the overflowing contents of the upper blind esophageal pouch. An unbelievably large amount of secretions comes from this pouch after its blind end has been exteriorized and opened and one can then understand how readily the infant can either drown itself or develop pneumonitis by aspirating the secretions which *overflow* from this segment. Therefore, the first operation, in our opinion, should be the exteriorization of the upper blind esophageal segment.

Under novocain—1:100—anesthesia an incision is made along the anterior border of the left sternomastoid muscle, the neurovascular bundle is retracted laterally, and the thyroid gland medially to expose the esophagus in the neck. The upper segment of the deformed esophagus is usually found to be much dilated; it is freed and cotton tape is passed around it. By traction on the tape and by blunt dissection, this segment of the esophagus can be freed completely and brought out through the wound in the neck. The segment shortens remarkably after it has been freed, so that it cannot be brought out onto the neck far from the working incision and in some instances it barely reaches the skin of the incision. The neck muscles are sutured around the esophageal segment with interrupted catgut sutures, and the skin is sutured with fine silk. Strips of collodion gauze are built up around the exteriorized esophageal stump, and, when they have become hard, the stump is split and held widely open with 4 traction sutures which are imbedded in the collodion (Figs. 2, 3, and 4).

In from 24 to 48 hours the second stage of the operation is performed. Under abdominal wall block, 1:100 novocain, a high left rectus incision is made up to the costal margin. The large fragile liver is carefully and gently retracted, and the esophageal hiatus is exposed. By downward traction on the stomach, the cardiac end of the stomach and the esophageal hiatus can be well exposed. An incision is made through the peritoneum around the hiatus and by blunt dissection the terminal esophagus is freed and pulled down into the abdomen (Fig. 5, frontispiece). Long narrow retractors are placed on the edges of the rent in the diaphragm and the dissection of the intrathoracic portion of the esophagus continued as far upward as possible. In 1 case our 1.5 inches of the esophagus were freed. A stout ligature of double medium silk is passed around the esophagus on an aneurysm needle, and the esophagus is ligated as tightly and as high as possible (Fig. 6, frontispiece). A second ligature is placed below the first and the esophagus is divided. The last ligature is left long and by traction on it to the left, the lesser curvature is thrown into relief and is ligated and divided about half way down its extent (Fig. 7, frontispiece). Thus the upper half of the stomach is mobilized. The skin on the upper left side of the rectus incision is now undermined to slightly above the costal margin and retracted. An incision is made through the muscle and peritoneum just below the costal margin and the upper end of the stomach with the attached portion of the esophagus is delivered through this incision (Fig. 8). A short transverse incision is made through the skin of the chest wall at the point to which it is estimated that the esophagus and stomach will reach. A subcutaneous tunnel is now made from this incision to the undermined skin flap at the costal margin. The stomach and esophageal stump are now drawn through this tunnel, and the skin edges of the thoracic wall incision are sutured around the esophageal stump (Fig. 8). The abdominal wound is closed in layers with fine silk. The esophageal stump which acts as a gastrostomy tube is opened after 24 hours.

By this operation access to the stomach is via a portion of esophagus which is advantageous in that no actual gastrostomy operation (Witzel, Frank, Janeway) has to be made, the natural relation between esophagus and cardiac end of the stomach is undisturbed, so that leakage of stomach secretions is prevented by the intact musculature at the cardia, by a sufficient length of esophagus, and by the valve-like action of the costal margin A great advantage is that the gastric stoma is placed high up on the thoracic wall—fourth rib in this case which has been reported—so that there remains only a short gap to be subsequently bridged by means of a skin tube.

The obvious defect in the operation is the fact that a blind esophageal pouch of varying length is left in communication with the trachea. This criticism can only be answered by the fact that in the 1 case herewith reported, there was no evidence during the postoperative course of any spilling over of secretions into the bronchial tree. The patient did die of pneumonia, but clinically this came on as an upper respiratory infection with red throat and with otitis media 42 days after operation. The ideal site of ligation of the esophagus would be very close to the point of fistula into the trachea, so as to leave remaining only as short a blind pouch as possible. Having had the experience with 1 case I believe that a much higher ligation of the esophagus can be achieved at subsequent attempts.

Before the reported operation was performed it had been decided to place a drain into the esophageal hiatus up to the ligated end of the esophageal stump for fear of leakage at the point of ligature. At the operation, however, the esophagus was so small and the danger of infection with the drain was considered so real that no drain was used. The fate of the ligated end of the esophagus caused much speculation and when the pneumonia and empyema came on 50 days after operation, it was suspected that the esophageal stump had finally leaked. At autopsy, however, the esophageal stump was found to be well closed by the ligature which was in place and as far as could be ascertained the esophagus was firmly embedded in scar tissue. It must be remembered, also, that this patient lived 62 days after operation. If leakage was to occur it should have been evident before the expiration of such a length of time. The danger of leakage from the ligated esophageal stump appears to be the greatest danger from the operation and is the weak spot in this operative procedure. It must be borne in mind, however, that there should be little strain on such a ligature since the esophagus above it carries no food, is empty save for some secretion from its mucosa, and has an outlet into the trachea. All that can be said is that in this 1 case there was no leakage and at the end of 62 days the stump appeared well healed, and there was no evidence of infection about it.

CASE REPORT

A 3-day old male infant was admitted to the Children's Hospital (Case 30597) on October 14, 1940. Soon after birth he was noted to have a great deal of mucus in his throat, and at the first feeding became cyanotic dyspneic, and nearly expired. An attempt was made to feed him by gavage, but the catheter would pass only a short distance down the esophagus. Barium was introduced by his pediatrician through the catheter and it was shown by x-ray that the esophagus ended in a blind pouch just within the thorax. He was referred to hospital.

His family history was of no significance save that his mother had a threatened abortion at about 2 months and was kept in bed for 6 weeks.

The baby was vigorous, of good development, and presented no evidence of other congenital defects. Aside from many coarse râles in both lungs his physical examination was negative. He was constantly coughing and sputtering saliva. The x-ray films which accompanied him showed that a blind pouch represented the upper portion of the esophagus and that the stomach was distended with air. It was obvious that he had the common type of congenital atresia of the esophagus in which its lower segment communicated with the trachea.

He was given 35 cubic centimeters of citrated blood and 40 cubic centimeters of 5 per cent glucose in normal saline solution intravenously. This was followed by 100 cubic centimeters of 5 per cent glucose in normal saline solution under the skin. The throat was kept free of secretions by aspiration and after being in the hospital 6 hours he was operated upon. Under local anesthesia, 1:100 novocain, the blind upper segment of the esophagus was brought out in the neck, surrounded by collodion gauze, and opened (Figs. 2, 3, 4). Much saliva drained through the opening and the coughing and sputtering ceased.

The next day (October 16, 1940) the procedure described and shown in Figures 5, 6, 7, and 8 was carried out under abdominal wall block, 1:100 novocain. The procedure was well tolerated. He received 40 cubic centimeters of citrated blood after operation and his water balance was kept up by intravenous and subcutaneous injections of 5 per cent glucose in normal saline solution.

On the day following operation a small catheter was inserted into the lower esophageal stoma and 5 cubic centimeters of formula—milk 1: water 3—was introduced through it into the stomach. The amount of fluid introduced into the stomach was steadily increased up to 45 cubic centimeters of formula. Like amounts of water were given as indicated to keep up the necessary fluid balance. The wounds healed well and by the twelfth day both wounds were entirely healed and the esophageal stomas were flush with the skin. The lower stoma,

which communicated with the stomach, showed little irritation around it at any time, but the constant flow of saliva from the upper stoma caused some skin damage, necessitating the frequent painting of the skin with compound tincture of benzoin.

There was a steady gain in weight from 6 pounds, 4 ounces immediately after operation to 7 pounds, 5½ ounces, 46 days later at which time an upper respiratory infection had become severe and finally terminated in pneumonia. After the first 2 weeks the child was bottle fed, a catheter was inserted into the upper stoma and the swallowed milk recovered. This was then introduced through the lower stoma into the stomach (Fig. 9). Several times the two stomas were connected by means of a rubber tube and the food was seen to flow rapidly down the tube into the stomach. It was not unusual for the infant to swallow without leakage in this way 2 ounces of formula in 2 minutes.

The convalescence was very gratifying and uncomplicated for 42 days. During this time there was no cough and no evidence of respiratory distress at any time save once or twice when the upper stoma became plugged by the catheter and there was coughing and sputtering when the feeding was given. Serious consideration was being given to connecting the two stomas by a skin tube when the patient developed an upper respiratory infection as evidenced by nasal discharge and catarrhal otitis media. This condition persisted for a week, at the end of which time a frank pneumonia developed. Within 48 hours an empyema was present and a few hours later a bronchial fistula with tension pneumothorax was evident. A closed thoracotomy was performed, sulfathiazole and blood transfusions were given and the patient was placed in an oxygen tent. He rallied considerably after a day or two and great hopes were had for his recovery. The pneumonia spread to the other lung and he died on December 17, 62 days after the completed operation.

An autopsy was performed. The abdomen was entirely negative. The portions of the stomach and esophagus which had been brought out under the skin of the thorax were intact and looked normal. The esophageal stump which had been brought out in the neck was likewise normal. There was a healing empyema cavity in the right pleural cavity. The right lung was practically entirely involved in a pneumonia process and there was a large opening through the lung tissue which communicated with a bronchus. The left lung was involved in a patchy bronchopneumonia throughout about one-third of its extent. The mediastinum was intact and there was no evidence of infection in it. The lower segment of the esophagus was not found to be dilated, it was empty, and communicated with the trachea as shown in Figure 10. Its blind end was well closed by the black silk ligature and by scar tissue and there was no evidence of leakage from it.

There was little shock connected with the operations which were readily done under local anesthesia. The lower esophageal stoma which communicated with the stomach functioned well in that there was little leakage and consequently little excoriation of the skin. This was probably due to the intact musculature at the esophagogastric junction and to the valve-like action of the costal margin. Peristaltic waves could be seen in that portion of the stomach which had been placed beneath the skin. There was no sign that the short pouch which was still communicating with the trachea was spilling secretions into the bronchial tree in amounts large enough to give symptoms. There was no leakage from the ligated esophagus 62 days after ligation. The cause of the pneumonia cannot be definitely stated, though the clinical picture was that of an upper respiratory infection progressing to a pneumonia rather than to a pneumonia secondary to aspiration of material from the connecting esophageal pouch.

SUMMARY

1. A type of operation for the cure of congenital atresia of the esophagus is described in which the proximal segment of the esophagus is brought out in the neck, the distal segment of the esophagus is ligated in the mediastinum as close to the communication with the trachea as possible, divided, and the portion of the esophagus attached to the stomach is brought out as a "gastrostomy tube."

2. The advantages of the operation described are that (1) an ideal type of gastrostomy is provided by using the lower end of the esophagus as a gastrostomy tube and (2) the distance between the two esophageal stomas is short so that they could be readily joined by a skin tube.

3. In the case reported simple ligation of the esophagus with silk did not result in leakage during the 62 days patient survived.

4. The 1 patient operated upon lived for 62 days and died of pneumonia which did not appear to result as a complication of the operation.

REFERENCES

1. Gage, M., and Ochsner, A. Ann. Surg., 1936, 103: 725.
2. Gamble, H. A. Ann. Surg., 1938, 107: 701.
3. Lanman, Thomas H. Arch. Surg., 1940, 41: 1060.
4. Leven, N. L. J. Thoracic Surg., 1936, 6: 30.
5. Richter, H. M. Surg., Gynec. & Obst., 1913, 17: 397.

A STUDY OF THE PLATELET COUNT AND THE COAGULATION TIME OF PLASMA AND WHOLE BLOOD FOLLOWING OPERATION

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THAT platelets influence coagulation is seemingly established, but whether they are essential to coagulation is still a question. Marked increases or decreases in the number of platelets shorten or lengthen the clotting time, but variations of 200,000 per cubic millimeter more or less, from a presumed normal of about 300,000, appear to make little difference in the coagulation time of the blood in the average, *normal* individual. The elusive character of blood platelets makes it difficult to define a normal numerical level. So many factors influence the number of blood platelets that it becomes difficult to be sure when a count is definitely normal.

In a very exhaustive review of blood platelets in health and disease, Tocantins calls attention to a number of commonplace factors which sharply alter the numerical level of platelets. Violent exercise, he says, will increase the platelet count from 18 to 180 per cent. Even changes in position from horizontal to vertical or vice versa will increase or decrease the count from 20 to 82 per cent. Furthermore, in the winter the counts in both the arterial and venous blood of man are significantly higher than in the spring, and in the summer there is a further decrease in number. Normal menses is associated with a decrease in platelets from 50 to 75 per cent during the first day of menstruation. A slow return to normal occurs during the remainder of the period.

Studies of blood platelets in postoperative patients have been made, and again variable results have been reported. Hueck and Dawbarn, Earlam, and Evans found a postoperative rise in the platelet count from the sixth to the tenth day. Allen found no significant or uniform variations in the platelet counts or the coagulation time following operation. Rosenbaum failed to observe thrombocytosis in women following thyroid operations. Olef found a drop in platelets during the first 24 hours following operation and a subsequent rise attaining a maximum at 14 days. From a differential study he determined that the increase in numbers was primarily of the small forms.

In 1936 Leslie and Sanford published a method of quantitative and qualitative estimation of platelets in their own plasma. They not only made direct counts of the platelets, but also studied the clotting time of platelet containing plasma and platelet free plasma. The former they labeled plasma A, the latter plasma B. The object of their method is to reduce to a minimum the breaking down of platelets caused by contact with glass, anticoagulants, and tissue juices.

It seemed worth while to utilize this method not only of counting the platelets but also of determining the coagulation time of platelet containing plasma and platelet free plasma in patients after operation. To this was added a record of the coagulation time of whole blood. This report is based on a study of 52 patients who underwent various major surgical procedures, such as: stomach, colon, and rectum resections, prostatectomies, hysterectomies, breast amputations, herniotomies, appendectomies.

METHOD

A brief description of the procedure published by Leslie and Sanford and since modified by Sanford follows.

The blood is taken from a vein through a fine needle into an iced and oiled syringe and is immediately transferred to an iced, paraffin

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TABLE I.—AVERAGE COUNTS

Date	Platelet count	Coagulation time in minutes: Plasma A	Plasma B	Whole blood
Preoperative.....	275,000	7.5	7.	7.5
1st postoperative day.	260,000	7.1	6.6	7.1
3rd postoperative day.	260,000	7.7	7.	7.6
5th postoperative day.	278,000	7.6	6.9	7.4
7th postoperative day.	292,000	7.6	7.	7.6
10th postoperative day.	301,000	7.6	7.	7.7
14th postoperative day.	289,000	7.6	7.2	7.3

coated, hematocrit tube. (A few drops of whole blood are drawn into a capillary pipette for determination of its coagulation time at room temperature.) The tube is packed in ice in a large centrifuge cup and centrifuged at 2000 revolutions for 1½ minutes. This will throw down the red and white cells but very few of the platelets. A few drops of the platelet containing serum are drawn up into a capillary pipette and its coagulation time at room temperature is determined. A few crystals of heparin are placed in a watch crystal resting on ice, and to these a few drops of the centrifuged plasma are added. After thorough mixing, this plasma is drawn up into a chilled pipette and introduced into a chilled counting chamber resting on ice. After 15 minutes allowed for settling, the count is made under high power. The balance of the plasma is again packed in ice and centrifuged at high speed for 15 minutes. The coagulation time of this plasma, now platelet free, is determined.

RESULTS

Fifty-two patients were satisfactorily studied. Estimations were made before operation, and in so far as possible, every day or every other day following operation until the patient's discharge from the hospital.

TABLE II.—AVERAGE COUNTS, CHOLECYSTECTOMY, MRS. D., AGED 43

Date	Platelet count	Coagulation time in minutes: Plasma A	Plasma B	Whole blood
April 6 preoperative....	260,000	7	9	7
April 9 postoperative...	240,000	7	4	8
April 11 postoperative..	182,000	7	6	7
April 12 postoperative..	234,000	6	4	5
April 13 postoperative..	164,000	9	7	8
April 15 postoperative..	305,000	7	6	7
April 17 postoperative..	269,000	6	6	6
April 19 postoperative..	345,000	7	5	4
April 22 postoperative..	216,000	6	6	lost
April 24 postoperative..	359,000	11	9	11

TABLE III.—AVERAGE COUNTS, GASTRIC RESECTION, MRS. C., AGED 41

Date	Platelet count	Coagulation time in minutes: Plasma A	Plasma B	Whole blood
April 16 preoperative...	354,000	6	6	6
April 16 immediately postoperative.....	352,000	6	6	8
April 17 postoperative...	310,000	6	5	6
April 18 postoperative...	320,000	6	6	8
April 20 postoperative...	320,000	7	6	7
April 23 postoperative...	428,000	9	9	10
April 25 postoperative...	350,000	7	7	8
April 27 postoperative...	390,000	6	6	7
April 30 postoperative...	268,000	5	5	5

The average preoperative platelet count was 275,000 per cubic millimeter (Table I). On the first postoperative day the count fell to 260,000 and remained at that level to the third postoperative day. On the fifth day the count rose to 278,000, on the seventh to 292,000 and on the tenth, to 301,000. By the fourteenth day in 24 patients who remained in the hospital it fell to 289,000.

The average coagulation time of the platelet containing plasma A was constantly practically the same as that of whole blood (Table I). Except for a slight drop on the first postoperative day neither varied much at any time. The reason that the coagulation time of serum and blood in this study was longer than the ordinarily accepted normal found in the laboratory lies in the fact that we dealt with chilled blood.

The average coagulation time of platelet free plasma B was constantly slightly less than that of plasma A. This may be due to the breaking down of the platelets during centrifuging, or may be due simply to the lapse of 15 minutes required for centrifuging.

TABLE IV.—AVERAGE COUNTS, BILATERAL HERNIORRHAPHY, MR. B., AGED 50

Date	Platelet count	Coagulation time in minutes: Plasma A	Plasma B	Whole blood
February 18 preoperative...	120,000	8	9	lost
February 19 postoperative.	120,000	9	8	lost
February 20 postoperative.	212,000	8	7	10
February 21 postoperative.	180,000	7	7	10
February 22 postoperative..	150,000	6	6	8
February 23 postoperative..	150,000	10	9	10
February 24 postoperative..	184,000	9	8	8
February 27 postoperative..	272,000	9	9	11
March 4 postoperative.....	120,000	7	9	10

A few patients had platelet counts which constantly ranged from 100,000 to 150,000, while others had persistent counts of 350,000 to 400,000. In a third group the individual counts varied from 150,000 to 400,000, from day to day. There was no discernible relationship between the platelet count and the time of coagulation. A platelet count of 120,000 was apt to be associated with a coagulation time of 5 minutes, and a count of 400,000 with a coagulation time of 10 minutes; the reverse was just as apt to obtain. We tried hard to establish a relationship between the count and the coagulation time but could find none.

One patient in this group developed thrombophlebitis the day after he was operated upon for intestinal obstruction. His platelet count ranged from 100,000 to 200,000, and coagulation time of his blood and serum was near the average of the other patients.

The platelet counts and coagulation times of 3 typical patients are recorded in detail to illustrate the variations from day to day (Tables II, III, and IV). It should be mentioned that the technique was mastered before the study was begun and was unchanged thereafter. Practically all of the studies were made during the forenoons of the late winter and spring months.

CONCLUSIONS

There is a 6 per cent drop from the preoperative level in the average blood platelet count on the first to third postoperative days. This is followed by a slow rise to 9 per cent above the preoperative level on the tenth day following operation.

The preoperative and the postoperative coagulation time of serum and of whole blood remained practically constant with the exception of a very slight drop on the first postoperative day.

In view of the average minimal variations in platelet count and coagulation time in patients who have undergone major surgical procedures, it seems doubtful whether these factors in themselves are of any significance in the development of postoperative thrombosis or embolism.

REFERENCES

1. Allen, E. V. Arch. Surg., 1927, 15: 254–264.
2. Dawbarn, R. Y., Earlam, R., and Evans, W. H. J. Path. & Bacteriol., 1928, 31: 833–873.
3. Hueck, H. Muenchen. med. Wchnschr., 1926, 73: 173.
4. Leslie, E. I., and Sanford, H. N. Am. J. Dis. Child., 1936, 51: 590–593.
5. Olef, I. Arch. Int. Med., 1936, 57: 1163–1185.
6. Rosenbaum, B. M. Zentralbl. f. Chir., 1924, 51: 2580–2582.
7. Sanford, H. N. J. Lab. & Clin. Med. (in press).
8. Tocantins, L. M. Medicine, 1938, 17: 155–258.

OXYGEN THERAPY IN SHOCK DUE TO HEMORRHAGE

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SHOCK produced by hemorrhage results in an anemic anoxemia due to loss of oxygen carrying hemoglobin and also a stagnant anoxemia due to the fall in blood pressure and a decrease in the volume and rate of blood flow.

Wood, Mason, and Blalock have recently reported their results of experiments upon 5 dogs. Shock following the blood loss of 2 per cent of the body weight by repeated hemorrhage produced a depression of the oxygen content of the arterial and venous blood. The blood pressure fell 55 millimeters of mercury and the hematocrit increased from 45 to 47 volumes per cent. After shock had developed, the inhalation of 100 per cent oxygen for only 15 minutes increased the arterial and venous blood oxygen content and the blood pressure rose 6 millimeters of mercury. The effect of more prolonged oxygen therapy was not reported.

Our work was done to determine if the continuous inhalation of high oxygen concentrations had a prolonged beneficial action, increased the tolerance to a greater volume of blood loss, and increased the duration of life of the animals.

METHODS

These experiments were done upon 20 normal dogs. The animals were deprived of food and water for 12 hours before they were used. One intravenous injection of sodium pentobarbital (25 mgm. per kilogram of body weight) was sufficient to maintain light anesthesia for the duration of the experiment. The left carotid artery was cannulated and attached to a mercury manometer. The dogs were bled ½ per cent of their body weight from a cannulated left fermoral artery every 15 minutes. When the blood pressure fell below 40 millimeters of mercury, the bleeding was reduced to ¼ per cent of the body weight every 15 minutes. Bleeding was discontinued when the blood pressure fell below 20 millimeters of mercury. Observations of the blood pressure, pulse, and respiration were made every half hour, and the total volume of blood lost and the length of life of each dog was noted. Capillary tube hematocrit determinations were made every hour upon 1 cubic centimeter samples of oxalated blood drawn from the right femoral vein.

Ten of the control dogs breathed atmospheric air (almost 21 per cent oxygen). The 10 treated dogs inhaled 100 per cent oxygen from a Heidbrink gas machine attached to a tracheal cannula.

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RESULTS

Loss of 2 per cent of the body weight in 1 hour by the control group of 10 dogs (Table I) resulted in an average increased hematocrit reading from 42 to 47 volumes per cent. The blood pressure fell from an initial average of 129 millimeters to 75 millimeters of mercury (fall of 54 mm.). The average pulse rate slowed from 153 to 147 per minute and the respiratory rate increased from 10 to 21 per minute. An average of the 10 dogs treated with 100 per cent oxygen (Table II) shows that following the loss of a similar volume of blood the hematocrit increased from 40 to 44 volumes per cent. The blood pressure fell from 137 millimeters to 102 millimeters of mercury (fall of only 35 mm.). The pulse slowed from 153 to 146 per minute, and the respiration increased from 21 to 37.

A blood loss of 4 per cent of the body weight over a period of 2 hours resulted in an average blood pressure of 35 millimeters in the control dogs and 39 millimeters of mercury in the dogs treated with oxygen. The hematocrit was 45 volumes per cent in both groups of dogs. The pulse and respiration was 154 and 35, respectively, in the control dogs and 139 and 39, respectively, in the treated group.

The group of dogs treated with oxygen tolerated a 15 per cent greater volume of blood loss (4.82 per cent of the body weight)

TABLE I.—SHOCK PRODUCED BY REPEATED SMALL HEMORRHAGE IN NEMBUTALIZED DOGS BREATHING ATMOSPHERIC OXYGEN

Dog	Normal				Bled 2 per cent body weight in 1 hour				Bled 4 per cent body weight in 2 hours				Total blood volume lost in per cent of body weight	Length of life in hours
	Hematocrit	Blood pressure	Pulse	Respiration	Hematocrit	Blood pressure	Pulse	Respiration	Hematocrit	Blood pressure	Pulse	Respiration		
1	35	120	128	6	35	100	189	12	35	40	140	36	4.0	4.00
2	36	145	184	12	60	22	152	52	60	20	150	60	3.0	2.75
3	36	130	152	20	39	105	148	28	40	60	136	72	6.0	3.50
4	38	100	140	10	51	38	124	24	D				2.4	1.83
5	40	120	176	12	38	60	148	32	30	20	200	40	4.5	2.75
6	42	110	136	12	46	100	100	20	42	30	100	28	5.4	3.75
7	45	130	124	4	52	40	144	10	57	30	116	38	3.1	2.50
8	50	118	160	12	52	90	168	12	43	50	204	16	5.0	2.75
9	52	140	170	6.	51	80	172	8	51	40	164	10	4.5	3.75
10	47	180	160	12	47	120	132	12	52	30	180	22	4.0	3.25
Av.	42	129	153	10	47	75	147	21	45	35	154	35	4.19	3.08

than the control dogs (4.19 per cent of the body weight). The average life of the dogs breathing atmospheric air was 3.08 hours and that of the dogs treated with oxygen was 3.60 hours (17 per cent longer).

EVALUATION OF RESULTS

The series of 10 dogs in each group was sufficiently large to compensate for the individual variations observed. The average initial hematocrit of the control dogs was 2 volumes per cent higher than the treated dogs. Although this is not a great variation in the 2 groups, the control group which had a higher initial hematocrit value could be expected to tolerate a greater loss of red blood cells by hemorrhage than the treated group of dogs with the lower hematocrit. In spite of this, a blood loss of 2 per cent of the body weight produced a fall in the blood pressure of 54 millimeters of mercury in the control dogs and only 35 millimeters in the dogs

TABLE II.—EFFECT OF INHALATION OF 100 PER CENT OXYGEN UPON SHOCK PRODUCED BY HEMORRHAGE IN NEMBUTALIZED DOGS

Dog	Normal				Bled 2 per cent body weight in 1 hour				Bled 4 per cent body weight in 2 hours				Total blood volume lost in per cent of body weight	Length of life in hours
	Hematocrit	Blood pressure	Pulse	Respiration	Hematocrit	Blood pressure	Pulse	Respiration	Hematocrit	Blood pressure	Pulse	Respiration		
1	28	130	140	16	33	120	164	28	38	40	148	36	5.5	3.75
2	36	136	160	8	39	80	140	40	41	34	148	56	4.5	3.00
3	38	150	168	16	51	140	180	60	49	50	144	16	5.1	4.33
4	38	140	144	12	40	110	160	16	37	35	154	48	4.7	3.75
5	40	130	160	20	43	90	168	20	44	40	180	36	4.7	3.25
6	40	140	144	16	42	118	144	20	35	30	100	28	5.2	4.08
7	42	120	160	16	48	80	140	38	54	20	140	26	4.0	3.25
8	42	150	180	28	42	100	144	64	41	42	164	68	4.6	3.25
9	46	130	120	68	49	50	88	44	50	42	100	40	4.4	3.75
10	48	150	160	16	52	140	140	40	60	65	118	40	5.5	3.66
Av.	40	137	153	21	44	102	146	37	45	39	139	39	4.82	3.60

treated with oxygen. After a blood loss of 4 per cent of the body weight over a period of 2 hours the blood pressure was 35 millimeters in the control dogs and 39 millimeters in the treated group. This demonstrates that oxygen therapy exerts a prolonged beneficial action upon the blood pressure. Anoxemia deprives the body of the important carotid sinus mechanism for maintaining the blood pressure. Gellhorn and Lambert reported that anoxemia greatly reduced the pressor reflexes of the carotid sinus in narcotized dogs. In addition anoxemia may cause a decreased blood volume by a loss of circulating fluids and protein through the anoxic capillary wall into the tissues. Landis has reported that the anoxic capillary wall of the frog's mesentery failed to retain plasma proteins and the permeability to fluid was increased 3 times normal.

Although theoretically one might expect a decrease in the hematocrit values following hemorrhage, an increased reading was observed in the majority of our dogs. Compensatory contraction of the liver, spleen, and areas of the capillary bed, press stagnant red blood cells into the circulation. This, augmented by loss of circulating fluid and protein through the anoxic capillary wall into the tissues, makes possible an increased, instead of a decreased, hematocrit.

Oxygen therapy in shock increases the oxygen content of the arterial and venous blood and therefore the amount available to the tissues. Our dogs treated with 100 per cent oxygen inhalation accordingly tolerated a 15 per cent greater blood loss and lived 17 per cent longer than the control dogs.

CONCLUSIONS

1. Inhalation of 100 per cent oxygen exerted a beneficial action upon shock produced by repeated small hemorrhage in dogs.

2. A blood loss of 2 per cent of the body weight in 10 control dogs produced a fall of 54 millimeters in the average blood pressure as compared to a fall of 35 millimeters of mercury of the 10 dogs treated with oxygen.

3. A blood loss of 4 per cent of the body weight over a period of 2 hours resulted in an average blood pressure of 35 millimeters in the control dogs and 39 millimeters in the treated dogs.

4. Hemorrhage in both groups increased the hematocrit values by 4 and 5 volumes per cent above normal.

5. The treated dogs had a slower pulse and a faster respiration than the control dogs.

6. The dogs treated by 100 per cent oxygen inhalation not only tolerated a 15 per cent greater blood loss (4.82 per cent body weight) than the control dogs (4.19 per cent of body weight) but also lived 17 per cent longer (3.60 hours) than the controls (3.08 hours).

7. Oxygen inhalation is a valuable adjunct in the treatment of shock following hemorrhage.

REFERENCES

1. Gellhorn, E., and Lambert E. Proc. Soc. Exper. Biol. & Med., 1938, 38: 315.
2. Landis, E. M. Am. J. Med. Sc., 1937, 193: 297.
3. Wood, G. O., Mason, M. F., and Blalock, A. Surgery, 1940, 8: 247.

THE UTERINE CONTRACTIONS OF EARLY PREGNANCY AND THEIR RELATION TO THE DURATION OF LABOR

A Study of 250 Patients Made with the Lóránd Tocograph

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IN a recent report (3) we noted certain changes in the uterine contractions which take place during the last 2 months of pregnancy. These included a progressive increase in the strength, duration, and frequency of the contractions, and also an increase in the tension of the uterine wall.

To supplement that study we are now reporting upon uterine activity over a longer period of gestation. Both studies were undertaken in order to learn the character of the contractions which occur *before* labor, on the hypothesis that such knowledge might assist in a better understanding of the activity which takes place *during* labor.

Specifically the present report deals with: (a) the time at which the contractions can first be detected, (b) the influence of advancing pregnancy upon their occurrence, and (c) the relation between their first appearance and the length of labor.

MATERIALS AND METHODS

The data, collected between December 1939 and September 1940, were supplied by patients drawn from the Maternity Department of the Hospital of the University of Pennsylvania and the Sheltering Arms Home.

Uterine activity was recorded with the two most recent types of tocograph (1, 2), the observations being carried out under the following conditions: The patient rested in her bed for 5 minutes. The tocograph was then placed upon the most prominent part of her abdomen in the midline, where it was held in place by a webbing girdle. The recording period lasted 30 minutes on the average, during which time note was made of any voluntary movements which might have influenced the tracing.

After the patients were delivered, the records of those who had presented any evidence of cephalopelvic disproportion or who were delivered by cesarean section were eliminated from further consideration. The presence of (a) intermittent contractions, (b) rhythmicity of contractions, and (c) any increase in the tension of the uterine wall was then determined in the tracings of the remaining patients.

The duration of pregnancy at the time that the records were made was computed from the interval of time elapsing before delivery, on the assumption that pregnancy lasts 280 days. Fetal development and menstrual history likewise were taken into consideration.

RESULTS

A series of 343 tocographic records was secured from 250 patients. The number of tracings showing contractions, rhythmicity, and an increase in tension of the uterine wall, is shown in Table I, arranged according to the duration of pregnancy at the time that they were made. The number of tracings and individuals corresponds for any given period in all tables, i.e., if a patient supplied two or more tracings during any 1 month, only the first one was employed.

UTERINE ACTIVITY DURING PREGNANCY

The days in pregnancy when fetal movement and uterine activity were first noted among the 343 tracings are as follows: earliest tracing, 110th day of gestation, earliest fetal movement, 130th day; earliest uterine contractions, 166th day; earliest rhythmicity, 166th day; earliest increase in tension, 222nd day.

From the Gynecean Hospital Institute of Gynecologic Research, Department of Obstetrics and Gynecology, University of Pennsylvania.

TABLE I.—UTERINE ACTIVITY DURING PREGNANCY—343 TOCOGRAPHIC TRACINGS BETWEEN 110TH AND 279TH DAYS, INCLUSIVE

Duration of pregnancy	Tocographic records showing							
	Number	Contractions				Increase in tension	
		Present		Rhythmic			
Weeks, inc.		No.	Per cent	No.	Per cent	No.	Per cent
13–16	1	0	—	0	—	0	—
17–20	6	0	—	0	—	0	—
21–24	12	1	8.3	1	8.3	2	16.7
25–28	38	2	5.3	1	2.6	7	18.4
29–32	19	3	15.8	1	5.3	3	15.8
33–36	44	17	38.6	6	13.6	11	25.0
37–40	223	158	70.9	78	35.0	97	43.5
Totals	343	175	—	87	—	120	—

Note: (a) absence of activity prior to the 21st week, (b) its subsequent progressive increase, (c) its greatest increase about the 32nd week and (d) the lack of 100 per cent activity during the last 4 weeks of gestation.

Contractions were observed in 51 per cent of all tracings taken during pregnancy, an increase in tension in 35 per cent, and rhythmicity in 25 per cent. The incidence of contractions might have been greater had the periods of observation been longer than 30 minutes.

Contractions. Intermittent contractions were not recorded until the 166th day of gestation, which was 56 days after the first tracing was made, and 36 days after fetal movement first appeared. Subsequently, the contractions recurred with increasing frequency (Table I). This increase, however, was not uniform. Breaking down the figures in Table I for the 2 months, from the 29th to the 36th week of pregnancy, inclusive, we find the incidence of contractions from week to week as shown in Table II. The first significant increase in activity appeared during the 32nd week of gestation.

Although the incidence of contractions increased as pregnancy advanced, only 70.9 per cent of tracings recorded their presence during the last month, and only 84 per cent during the last week.

Rhythmicity. Rhythmicity appeared as early in pregnancy as contractions (Table I), but subsequently was observed less frequently. It increased in frequency also as pregnancy progressed, and like contractions, it increased

TABLE II.—INCIDENCE OF UTERINE CONTRACTIONS FROM 29TH TO 36TH WEEK OF GESTATION, INCLUSIVE

Week of pregnancy	Tracings showing contractions	
	Present No.	Absent No.
29	0	6
30	1	5
31	0	3
32	2	2
33	3	10
34	4	5
35	6	5
36	4	7

Note increase in number of tracings recording contractions beginning at the 32nd week.

at the greatest rate about the 32nd week of gestation. During the last month it appeared in only 35 per cent of tracings, and during the last week in only 60 per cent.

Increase in tension of the uterine wall. An appreciable increase in the tension of the uterine wall was first observed on the 222nd day of gestation, which was 56 days after uterine contractions were first noted. It also increased in frequency as pregnancy progressed, showing its greatest increase, like the two other characteristics, about the end of the 32nd week of gestation. During the last month it was present in only 43.5 per cent of tracings and during the last week in only 52.3 per cent.

TABLE III.—EFFECT OF CONTRACTIONS UPON THE DURATION OF LABOR

Duration of pregnancy	Contractions			
	Present		Absent	
	Tracings	Average duration of labor	Tracings	Average duration of labor
Weeks, inc.	No.	Hours	No.	Hours
13–16	0	—	1	8.0± 0.0
17–20	0	—	6	19.6±17.5
21–24	1	2.3± 0.0	11	19.0±16.3
25–28	2	6.6± 0.7	36	12.1±12.0
29–32	3	4.6± 2.4	16	15.2±15.1
33–36	17	14.4±13.5	27	12.9± 8.6
37–40	158	13.0± 9.7	65	11.2± 8.9
Totals	181	—	162	—

Note: (a) absence of contractions prior to 21st week, (b) small number of tracings made before the 33rd week of gestation which recorded the presence of contractions, and (c) the shortness of the labors of the individuals concerned.

TABLE IV.—EFFECT OF CONTRACTIONS UPON THE DURATION OF LABOR ACCORDING TO PARITY

Duration of pregnancy	Contractions							
	Present				Absent			
	Primigravidas		Multigravidas		Primigravidas		Multigravidas	
	Number	Labor	Number	Labor	Number	Labor	Number	Labor
Weeks, inclusive		Hours		Hours		Hours		Hours
13–16	0	—	0	—	1	8.0± 0.0	0	—
17–20	0	—	0	—	3	23.6± 19.1	3	15.6± 14.6
21–24	0	—	1	2.3± 0.0	8	18.4± 14.1	3	20.6± 21.0
25–28	1	5.9± 0.0	1	7.3± 0.0	15	19.2± 14.6	21	7.0± 5.4
29–32	2	3.3± 1.8	1	7.3± 0.0	11	19.6± 16.3	5	5.7± 3.3
Totals	3	—	3	—	38	—	32	—
Weighted average	—	4.2± 1.2	—	5.6± 0.0	—	19.2± 15.0	—	8.9± 7.4

Average duration of labor of patients traced prior to 33rd week of pregnancy, arranged according to parity. Note short duration of labors, both multigravidas and primigravidas, whose tracings recorded the presence of contractions.

UTERINE ACTIVITY IN RELATION TO DURATION OF LABOR

Effect of contractions. The relation between the occurrence of intermittent contractions and the duration of labor is summarized in Table III. If pregnancy is viewed as a whole, the weighted average duration of labor of individuals whose tracing showed contractions was the same as for the persons whose tracings showed no contractions. This statement likewise is true when considering the effect of the occurrence of contractions during the 33rd to the 40th weeks of pregnancy upon the duration of labor.

TABLE V.—DURATION OF LABOR OF PATIENTS STUDIED BEFORE 33RD WEEK OF PREGNANCY ACCORDING TO PARITY

Parity	Individuals		
	Contractions	Number	Average duration of labor
			Hours
All	Absent	70	14.4± 10.2
	Present	6	4.9± 0.6
			Difference 9.5± 9.6
			Standard error ± 1.2
Primigravidas	Absent	38	19.2± 15.0
	Present	3	4.2± 1.2
			Difference 15.0± 13.8
			Standard error ± 2.5
Multigravidas	Absent	32	8.9± 7.4
	Present	3	5.6± 0.0
			Difference 3.3± 7.4
			Standard error ± 1.3

Note that presence of contractions before 33rd week of gestation has resulted in labors significantly shorter than those of patients who did not exhibit contractions. The difference is more than three times the standard error in the combined group and in the primigravida group, and nearly that in the multigravida group.

Seventy-six tracings were secured prior to the 33rd week of pregnancy (Table III). Of these, only 6, or 7.9 per cent, recorded the occurrence of contractions; the latter tracings belonged to individuals whose average duration of labor was only 4.9 ± 1.6 hours, whereas the 70 tracings showing *no* contractions belonged to women whose average duration of labor was 14.5 ± 13.7 hours. The difference between these two figures is 9.6 hours and its standard error was found to be 3.1 hours, indicating that the difference is statistically significant.

The duration of labor of these patients in relation to their parity is summarized in Table IV, the totals of which table are compared in Table V. It will be noted in the latter table that the presence of contractions early in pregnancy influenced the duration of labor of the primigravidas to an extent which is statistically significant, and that of the multigravidas to practically the same extent. A difference of three times the standard error is considered significant.

Effect of rhythm and an increase in tension. The effect of rhythmicity and an increase in tension upon the duration of labor also was

determined. The early appearance of these characteristics had no effect upon the duration of labor.

OBSERVATIONS

The present study indicates that uterine contractions were first observed on the 166th day of gestation. This refers to spontaneously arising movements. Prior to this time we have noted contractions which arose as the result of fetal activity, but none that arose spontaneously. It is apparent, therefore, that the uterus does contract prior to the 166th day under stimulation by the fetus, and it is equally likely that it will contract before that time as the result of manual excitation. The fact, however, that we detected fetal activity considerably in advance of any spontaneously arising uterine activity, suggests that in all probability we have recorded some of the earliest uterine movements.

Although spontaneous uterine contractions begin about the 21st week of gestation, it is of interest that this activity shows its greatest increase about the 32nd week. The reason for this is not known. That the sensitivity of the uterus increases markedly at this time is likewise shown by its response to pituitary extract (4). The administration of this drug at weekly intervals throughout pregnancy has shown that the majority of patients exhibit their first responses to it at this time.

The present study centers about the relation between early activity and the duration of labor. It seems apparent that the patient who experiences early activity is more than likely also to have a short labor. Such a sequence would seem to be a logical one. On the other hand, the patient who does not experience early activity also may have a short labor.

During the last 2 months of pregnancy (Table III), the mere presence of contractions appears to have no influence upon the duration of labor. Studies are in progress, however, for the purpose of determining whether the magnitude, or any other characteristic of the contractions occurring at this time have prognostic value regarding the nature of the labor.

SUMMARY AND CONCLUSIONS

1. The uterine contractions of 250 women were recorded between the 110th and 279th day of pregnancy with a Lóránd tocograph.

2. Although fetal movement was detected as early as the 130th day of gestation, no spontaneous uterine contraction was noted until the 166th day.

3. Uterine activity increased progressively during the remainder of pregnancy, its greatest increase in the middle of pregnancy taking place about the 32nd week.

4. Patients experiencing uterine contractions prior to the 33rd week of gestation experienced significantly shorter labors than those who did not have such activity.

5. The presence of uterine contractions after the 32nd week of pregnancy was of no value in predicting the character of the labor.

REFERENCES

1. Loewi, S. (Lóránd). Zentralbl. f. Gynaek., 1933, 57: 554–558.
2. Lóránd, S. (Loewi). Monatschr. f. Geburtsch u. Gynaek., 1936, 103: 137–145.
3. Murphy, D. P. Surg., Gynec. & Obst., 1940, 70: 129–135.
4. Idem. Surg., Gynec. & Obst., 1941, 73: 175.

LIVER INSUFFICIENCY IN TOXIC GOITER AND ITS TREATMENT

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PREOPERATIVE management of goiter patients has become a fairly standardized regimen embracing the discriminate use of iodine, a diet high in calories and carbohydrates, sedation, and enforced rest. During the past decade these principles have accomplished much toward lowering mortality and morbidity in thyrotoxic patients subjected to surgery. For all but a limited group of severely toxic patients the present day treatment, in the hands of an experienced surgeon, is entirely adequate. There remains, however, the not too occasional bad risk patient who enters the hospital in impending or actual thyroid crisis. Surgeons have learned that deferring surgery in these individuals reduces the incidence of postoperative complications and lowers postoperative mortality. Such judgment on the part of the surgeon improves operative statistics. It may also be a factor in explaining why, in many localities, thyroid crisis is now encountered more frequently before than after surgery.

More often than not these poor risk patients have carried their goiter a long time, have taken iodine over long periods, and have become refractive to its quieting influence at a time when iodine medication would accomplish the greatest benefit. Frequently, such patients are suffering not from so called hyperthyroidism associated with diffuse hyperplasia of the thyroid, but from a failing heart associated with a nodular, degenerated goiter of long standing. In some the situation is complicated by a toxic psychosis. These severely toxic patients invite continued interest in the pathological physiology of toxic goiter. Controlled observations have convinced us that certain specific therapeutic measures can reduce materially the preoperative and postoperative morbidity in extremely toxic goiter patients.

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LIVER DAMAGE IN THYROTOXICOSIS

The damaging effect of thyrotoxicosis on the liver has been appreciated by pathologists for years. Numerous reports on autopsy studies are in accord (3, 8, 30, 35, 41, 43). Both acute and chronic hepatic lesions are recognized, depending on the severity and duration of the thyrotoxic state. Changes ranging from mildly acute degenerative alterations, characterized by fatty metamorphosis and acute necrosis, to subacute toxic atrophy and cirrhosis have been described. The most extensive degenerative changes have been encountered in thyroid crisis, and a few authorities favor the idea that acute hepatic failure is the underlying basis of thyroid crisis (7, 11, 21).

Interpretation of pathological alterations in the liver of a patient subjected to extreme degrees of fever during thyroid crisis must be guarded. How can one decide in retrospect whether the disintegration of hepatic parenchyma produced, or was the result of, hyperpyrexia? Notwithstanding this controversial aspect, the fact remains that typical degenerative hepatic lesions have been found repeatedly in association with toxic goiter in which extreme degrees of fever was not a part of the clinical picture. Using microscopic sections of autopsy material as criteria, one can but speculate on the degree of impaired hepatic function before death. Beaver and Pemberton in an analysis of the hepatic changes in 107 cases of exophthalmic goiter, stated that the hepatic lesions were such that disturbed hepatic function would have been anticipated in 40 per cent of the cases. It seems probable that the functional state of the liver cell must be compromised appreciably long before the stage of outspoken degeneration is reached.

Within the past few years studies of liver function in goiter patients leave no doubt that measurable liver damage, frequently marked, exists in toxic goiter. Using the Quick hippuric

acid test, Bartels found that 130 of 148 cases of hyperthyroidism showed evidence of impaired hepatic function. Boyce and others (18, 27, 37) have also published data on impaired liver function in selected goiter patients; however, we are aware of no comprehensive study pertaining to the incidence of hepatic insufficiency in a significant number of unselected goiter patients. The present report is based on studies begun in November, 1939, on over 200 consecutive patients subjected to thyroidectomy.

LIVER FUNCTION TESTS

The ideal liver function test has not been discovered. This is so for several reasons. In the first place the liver possesses multifold functions and no single test could be expected to reveal the status of the organ as a whole. It is the only organ the removal of which causes unexplainable death. Second, the liver is a large organ possessing a tremendous factor of safety and ability to regenerate, facts which demand significant damage before a state of measurable hepatic insufficiency can be demonstrated. Furthermore, the liver is inaccessible; its rôle in metabolism can be investigated in patients only by indirect methods. Nevertheless, there are several liver function tests which are of value, particularly if their use is restricted to serial determinations during the course of disease or treatment. These same tests are of considerably less value when they are used solely for diagnostic purposes.

Various liver function tests are aimed at one of many and varied hepatic functions. Sugar tolerance tests are directed at the function of carbohydrate metabolism. Blood determinations for nonprotein nitrogenous constituents have to do with protein metabolism. The various dye elimination tests measure the ability of the Kupffer cells, as well as the rest of the lymphatic reticulo-endothelial system, to remove foreign substances injected into systemic circulation. The hippuric acid test, adapted by Quick (1933), is concerned with the hepatic functions of synthesis and detoxication.

The ability of the liver to detoxify injurious breakdown products arising in the course of normal metabolism or from infectious or disease processes is of primary importance. It is of especial interest to the surgeon who must frequently evaluate an ill patient as a surgical risk. Toxic substances are changed in the healthy liver to relatively inert products which are subsequently eliminated in the body excretions. These changes are accomplished by biochemical processes involving either oxidation, reduction, deamination, or conjugation. All are vital processes. When the detoxifying ability of the liver is impaired or overtaxed the body as a whole suffers from the resulting toxemia. One of the most common means of hepatic detoxication is the combination of toxic substances with the aminoacid glycine to form a less toxic compound. Glycine is synthesized by the hepatic cells, but the liver does not store it. Ordinarily there is also an exogenous supply of glycine in the protein of the diet. In the absence of exogenous glycine the liver manufactures this substance at a constant and fixed rate (27, 28). The hippuric acid test measures the ability of the liver to manufacture and combine glycine with ingested benzoic acid, itself nontoxic, to form hippuric acid. The hippuric acid is excreted by the kidneys. The validity of the hippuric acid test in various states of liver damage has been established by a number of investigators (2, 7, 27, 28, 29, 37). The test is simple, inexpensive, does not greatly inconvenience the patient, and can be reduplicated with remarkable accuracy in a given individual. Furthermore, it utilizes a normal mechanism and does not put a strain on a liver that may be damaged. Results of the test are not valid in the presence of (1) a pathological process which interferes with the emptying of the stomach or absorption from the intestine, or (2) advanced kidney disease with impaired urea clearance.

During the past year we have employed this and other liver function tests in over 500 medical and surgical patients with and without liver damage. From these data we have satisfied ourselves not only of the reliability of the hippuric acid test but have found it consistently sensitive to degrees of impaired hepatic function not reflected by certain other liver function tests. The test is carried out essentially as described by Quick (29) with several minor modifications:

TABLE I.—CLINICAL CLASSIFICATION OF GOITERS STUDIED

	No.	Total	Per cent
Nodular			
Nontoxic	10		
Toxic	75		
Fetal adenoma	38	123	59.4
Diffuse			
Colloid	14		
Hyperplastic	50		
Recurrent	14	78	37.6
Malignant		2	1.0
Hashimoto struma		4	1.9

One hour after a light breakfast of tea and toast the patient empties the bladder. This specimen is discarded. The test solution, consisting of 6 grams of sodium benzoate made up to 1 ounce with peppermint water, is given, followed by one-half glass of water. The drinking of water is discouraged but not prohibited during the next 4 hours. Four hours after the test solution is given the bladder is again emptied. If the patient has voided during the test period this specimen is included with the 4 hour specimen. It is imperative that all urine formed during the 4 hour period be collected and that the bladder be empty at the end of this period.

The 4 hour urine specimen is brought to a volume of 145 cubic centimeters. If the volume of the specimen is less than 145 cubic centimeters it is diluted to the specified volume with tap water. As a rule it is necessary to concentrate the specimen by evaporation on a steam bath. Before heating, several drops of glacial acetic acid are added to the urine.

After evaporation, the urine is cooled to room temperature, the final volume is corrected to 145 cubic centimeters and the specimen is transferred to a 250 cubic centimeter beaker of known weight. Then 5 cubic centimeters of concentrated hydrochloric acid is added and the specimen is stirred vigorously with a glass rod for several minutes to precipitate the hippuric acid completely. The acidity is tested with Congo red indicator paper. If the color does not change from red to blue more concentrated hydrochloric acid is added until this color change is produced.

The beaker is allowed to stand at room temperature for 30 minutes. The precipitate is then stirred and transferred to a moistened filter paper in a Buchner funnel and filtered by suction. The precipitate is washed once with a 20 cubic centimeter portion of cold water and air is allowed to be drawn, by suction through the precipitate for several minutes. The precipitate and filter paper are transferred back to the 250 cubic centimeter beaker and allowed to dry at room temperature 16 to 20 hours. The beaker and its contents are weighed.

Calculation: Weight of beaker and contents minus weight of beaker and paper plus 0.50 grams equals grams of hippuric acid excreted in 4 hours. The value 0.50 grams represents the amount of hippuric acid dissolved in 150 cubic centimeters of acidified urine. The purpose in making the volume to 150 cubic centimeters is to avoid variables which might be introduced by varying too greatly the volume from which the hippuric acid was precipitated.

We have found that 150 patients in whom there was no reason to suspect liver damage excreted from 4.5 to 5.5 grams of hippuric acid in 4 hours following the ingestion of 6 grams of sodium benzoate. We report hippuric acid values in terms of per cent, using 5 grams (average of 150 normal subjects) as 100 per cent. A value of 90 per cent is considered the lower limit of normal.

Having available a reliable means of estimating one clinically important phase of hepatic activity, we have attempted to evaluate, using quantitative data instead of clinical impressions, the efficacy of therapeutic measures directed at improving impaired liver function in goiter patients. Our observations demonstrate a striking physiological relationship between toxic goiter and the liver. The functional state of the liver appears to have a profound influence on postoperative morbidity, is in turn suppressed significantly by postoperative febrile states. Marked elevation of basal metabolic rate is frequently associated with impaired liver function; however, the greatest degrees of hepatic insufficiency were encountered in toxic patients with chronic thyroid disease in whom the basal metabolic rate was normal or only slightly elevated (apathetic hyperthyroidism of Lahey). Toxic psychosis in goiter patients, either before or following surgery, is preceded and accompanied by evidence of hepatic insufficiency. Finally, selection of anesthesia for thyroidectomy is of utmost importance if liver damage is to be minimized as a result of the operative procedure.

INCIDENCE OF LIVER INSUFFICIENCY IN THYROTOXICOSIS

This study is based on 207 consecutive, unselected goiter patients subjected to thyroidectomy. In Table I the patients are grouped according to the clinical and laboratory diagnosis. Approximately 60 per cent had nodular goiters, in 38 per cent the thyroid was diffusely enlarged, slightly less than 2 per cent presented Hashimoto struma, and in 2 instances (less than 1 per cent) malignancy was suspected and found.

Upon admission to the hospital 55 per cent of the group as a whole had subnormal liver

TABLE II.—INCIDENCE AND DEGREE OF LIVER INSUFFICIENCY IN GOITER IN 207 CONSECUTIVE CASES

Clinical classification	Per cent of series	Liver function									
		Normal		Impaired							
				Moderate		Marked		Severe		Extreme	
		100 per cent or over	90 to 100 per cent	80 to 90 per cent	70 to 80 per cent	60 to 70 per cent	50 to 60 per cent	40 to 50 per cent	30 to 40 per cent	20 to 30 per cent	10 to 20 per cent
Nodular nontoxic	4.7	80	20								
Toxic	36.2	23	15	29	21	4	8				
Fetal adenoma	18.3	36	21	21	21						
Diffuse colloid	6.8	27	38	7	21	7					
Hyperplastic	24.1	16	4	10	24	20	6	6	4	0	6
Recurrent	6.8	64	0	36							
Malignant	1.0	100									
Hashimoto struma	2.0	0	50	0	0	50					

function as measured by the hippuric acid test. The incidence of liver insufficiency, summarized in Table II according to clinical classification, is noteworthy because it reveals a fundamental finding, namely, that liver insufficiency occurs in *all* types of goiter except a few instances of nontoxic nodular goiter.

Toxic nodular goiter. In this group 38 per cent showed normal liver function, approximately 29 per cent had mild liver damage, while in the remaining third hepatic function was diminished significantly. Twelve per cent of the group had marked impairment.

Fetal adenoma. Approximately three-fourths of the patients in this group had normal or slightly impaired liver function. In the remaining 26 per cent moderately impaired values were obtained. In the majority of the latter the goiters were found to contain varying degrees of degenerative change.

Diffuse colloid. Nearly one-third of the patients in this group showed evidence of significant liver damage. At operation these goiters also presented varying degrees of degenerative change.

Diffuse hyperplastic goiter. The most severe liver damage was encountered in this group, over half having liver function under 80 per cent of normal. Forty per cent of the patients with diffuse toxic goiter had seriously impaired hepatic function (less than 60 per cent of normal). Three patients entered the hospital in thyroid crisis with liver function between 10 and 20 per cent of normal.

LIVER INSUFFICIENCY AND BASAL METABOLIC RATE

Correlation of liver damage in our goiter patients with the clinical history and basal metabolic rate reveals some interesting relationships. Duration and intensity of thyroid dysfunction rather than the type of goiter present seems the deciding factor in impaired liver function. This is especially true of patients with goiters of long duration who have had indeterminate weight loss and history suggesting many remissions and exacerbations of the thyrotoxic state. A high percentage of such patients had normal or slightly elevated basal metabolic rate. When liver function and basal metabolic rate are correlated one is forced to the conclusion that no constant relationship exists. More important, it becomes very evident that the basal metabolic rate can be dangerously misleading if it is used as a guide in evaluating a prospective surgical risk. We encountered extreme degrees of functional liver damage in patients with basal rates of plus 30 or less. The relationship between liver function and basal metabolic rate in 45 patients is

TABLE III.—EFFECT OF PREOPERATIVE TREATMENT ON LIVER FUNCTION IN TOXIC GOITER

Patient No.	Sex	Age	Clinical diagnosis	Admission		Preoperative		Operation	Postoperative		
				Basal metabolic rate	Per cent liver function	Days' treatment	Per cent liver function		Per cent 2 days	Per cent 8 days	Basal metabolic rate
A. Routine treatment											
5	F	55	Toxic nodular	+35	81	9	84	Bilateral	74	71	+26
10	F	37	Exophthalmic	+35	53	12	52	Lobectomy	50	71	+ 6
11	F	38	Toxic nodular	+36	82	7	88	Bilateral	81	77	+ 5
12	F	56	Fetal adenoma	+46	69	12	74	Lobectomy		69	−16
35	F	27	Exophthalmic	+85	64	9	85	Lobectomy		83	+63
38	F	47	Toxic nodular	+62	60	16	25	Lobectomy	*	63	+30
39	F	53	Toxic nodular	+ 9	79	4	84	Bilateral			−15
121	F	62	Degenerated colloid	+26	73	6	85	Lobectomy	56		+16
123	F	24	Exophthalmic	+61	60	10	15	Bilateral	Fatal crisis		
127	F	26	Toxic nodular	+43	71	5	92	Bilateral	70		−21
B. Insulin-glucose											
4	F	49	Exophthalmic	+61	67	4	94	Bilateral	66	75	+22
8	F	40	Diffuse toxic	+ 5	67	7	100	Bilateral		85	0
22	F	37	Diffuse toxic	+40	32	6	90	Bilateral	80	97	+15
49	F	27	Diffuse toxic	+71	62	5	85	Bilateral	80	85	+ 5
87	F	17	Exophthalmic	†	15	11	100	Bilateral	‡		+ 6
C. Liver extract—bile salts											
52	F	45	Diffuse toxic	+66	72	10	85	Bilateral	65	76	+17
63	F	49	Diffuse toxic	+47	80	4	95	Bilateral	82	93	+12
65	F	41	Nodular toxic	+10	73	5	100	Bilateral		90	
90	F	27	Diffuse toxic	+34	77	5	92	Bilateral	73		+10
91	F	38	Diffuse toxic	+45	96	3	100	Bilateral	84	100	− 4
117	F	25	Nodular toxic	+39	62	5	85	Bilateral	79	90	+ 4
145	F	39	Diffuse toxic	+80	74	8	97	Bilateral			
D. "Works"											
38	M	42	Exophthalmic	+43	30	5	79	Bilateral	64	96	
48	F	44	Nodular toxic	+23	76	5	100	Bilateral	98	100	0
88	F	44	Nodular toxic	+58	48	7	70	Lobectomy	54	85	−13
54	M	34	Diffuse toxic	+40	83	8	92	Bilateral	67	96	+ 5
89	F	72	Diffuse toxic	+22	15§	35	80	Lobectomy		58	−20
70	F	43	Diffuse toxic	+12	50	6	95	Bilateral			− 7
71	F	49	Diffuse toxic	+70	72	8	92	Lobectomy	83	100	+54
119	M	56	Diffuse toxic	+26	15‖	19	81	Lobectomy	77		− 9
128	F	50	Diffuse toxic	+56	63	12	100	Bilateral	94	100	− 7
138	F	47	Diffuse toxic	+51	76	11	97	Bilateral	83		
131	F	37	Diffuse toxic	+24	40	10	73	Lobectomy	62	71	− 2
136	M	59	Nodular toxic	+18	48	10	86	Bilateral		85	− 4
150	M	58	Diffuse toxic	+61	77	8	100	Bilateral	100		

A. Routine treatment: complete bed rest, sodium bromide 15 grains t.i.d., luminal if necessary, lugol solution, 15 drops t.i.d., vitamin B complex (betalin) 30 grains daily. B. Insulin-glucose: routine treatment plus insulin, 5 units t.i.d. a.c. 100 grams glucose intravenously with insulin, 20 units daily last 3 days before operation. C. Liver extract—bile salts. Routine treatment plus 45 grains liver extract (Wilson) and 30 grains bilron (Lilly) daily. D. "Works." All measures in A, B, and C plus glycine drams 6 daily.

*Extremely stormy postoperative course; see text. †Entered hospital in crisis. ‡Delayed recovery due to lobar pneumonia. §Developed toxic psychosis 3 days after admission. ‖Developed beginning crisis day after admission. Both responded to treatment.

shown graphically in Figure 1. If a direct relationship existed between severity of thyroid dysfunction as reflected by the basal metabolic rate and the degree of liver damage associated with thyrotoxicosis, one would expect moderate degrees of impaired function (70 to 80 per cent of normal) with moderately elevated basal rates (up to plus 40) and greatest liver damage with high basal metabolic rates. This is not the case, otherwise the values on the graph (Fig. 1) would fall within the diagonal lines drawn parallel on the graph. Less than half of the patients show such a relationship. On the other hand, the 9 patients represented in the lower left corner of the graph (20 per cent) show normal or moderately elevated basal rates with marked to extreme impaired liver function. This type of goiter patient requires more than ordinary preparation for surgery.

SPECIFIC MEASURES TO IMPROVE LIVER FUNCTION

Having demonstrated the presence of measurable hepatic insufficiency in more than half of goiter patients entering the hospital for surgery, several questions become pertinent. Foremost are: (1) How much does liver function improve under conventional treatment? (2) What is effect of thyroidectomy on liver function? (3) What specific measures promote greatest improvement in liver function?

Serial tests of liver function were made on 132 toxic goiter patients. These were made on admission, after a period of preoperative preparation (3 to 10 days or longer), 2 days after operation, and before dismissal, usually on the eighth day after operation. In the 75 remaining patients tests of liver function were made on admission and again before operation in instances in which the initial test was subnormal. Lack of space prevents the publication of the data on all of these patients. Presenting averages would show the general trend of results but would conceal many significant individual variations. Likewise, to submit only selected cases would lead to overemphasis in one direction or another. Consequently, we have sought to present typical cases in a manner that represents an unbiased cross-section of the group as a whole.

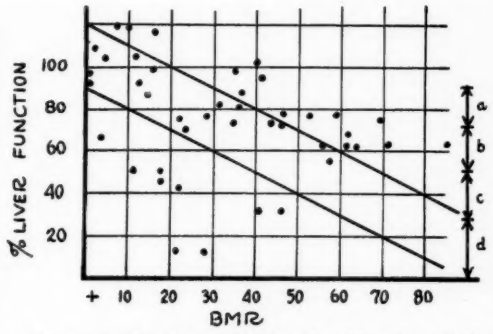

Fig. 1. Graph showing relationship of basal metabolic rate and hepatic insufficiency. *a*, moderate damage, *b*, marked damage, *c*, severe damage, *d*, extreme damage.

Control group. These patients received conventional preoperative treatment including complete bed rest, Lugol's solution, bromides or one of the barbiturates, and a high carbohydrate diet supplying between 3000 and 5000 calories daily. In addition, vitamin B complex (betalin compound, 30 grains daily) was included as part of the routine treatment inasmuch as this had been our practice for more than a year preceding the commencement of the present study.

"Treated" group. Together with the treatment outlined, specific measures to improve liver function were instituted. These included (a) intravenous glucose, with and without insulin; (b) insulin; (c) bile salts; (d) liver concentrate; and (e) glycine. These various therapeutic measures were used alone and in different combinations in an attempt to evaluate their individual and collective merits. Table III shows typical results on improved liver function obtained by the various procedures employed. We have concluded that maximum recovery of impaired liver function in toxic goiter is obtained in the shortest period by a regimen including all of the specific procedures listed, i.e. the whole is greater than the sum of its parts Noteworthy in this respect is the observation that in essentially all instances of toxic goiter the liver damage present is not irreparable. No instance was encountered in which impaired liver function was not greatly improved or normal following specific measures directed at restoring hepatic function. In contrast, a number of patients

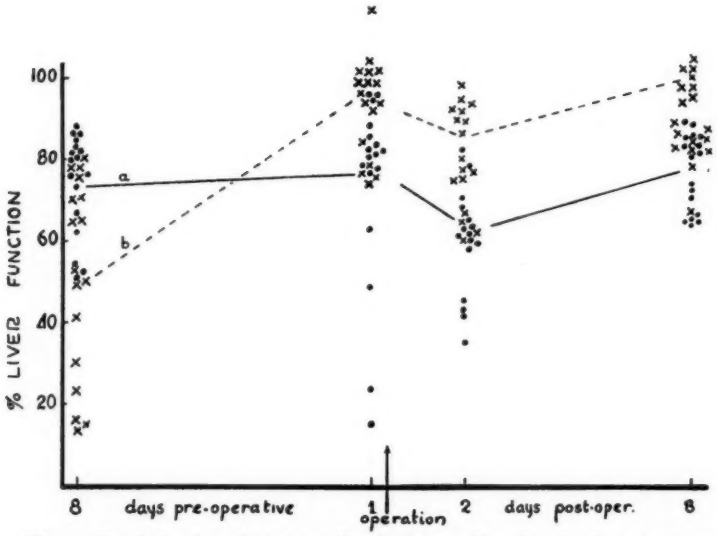

Fig. 2. Graph showing effect of specific measures on liver function in toxic goiter and the effect of thyroidectomy: *a*, routine treatment (———), *b*, routine treatment plus specific measures (x——x).

failed to show comparable improvement on conventional preoperative treatment.

Figure 2 represents a "spot" graph showing the response of patients in the control group compared with patients receiving additional therapeutic measures. Individual values are given to illustrate individual variations. Group averages are shown also. It will be noted that liver function in the control group ranged between 45 and 85 per cent (average 70 per cent) on admission whereas the treated group were far more toxic, showing values varying from 12 to 80 per cent of normal (average 50 per cent). At the end of the preoperative period the general trend in improved liver function approaches normal in the "treated" group. In the conventionally treated series the trend toward improved liver function in some is offset by subjects who showed no response or progressively impaired liver function on routine treatment.

The 2 individuals represented on the graph in Figure 2 with preoperative liver function of 15 and 25 per cent (also in Table III, Nos. 38 and 123) terminated abruptly the desire to secure further data on conventional treatment in extremely toxic goiter patients.

The former, No. 123, a female 24 years of age, had a toxic exophthalmic goiter. On admission liver function was 60 per cent of normal (basal metabolic rate plus 61). Response to routine treatment seemed satisfactory and at the end of 10 days the patient appeared clinically ready for surgery. When the result of the preoperative hippuric acid test was reported the evening before operation it was decided that there had been some error in the performance of the test. A check up in view of subsequent events proved that this was not the case. The standard test solution had been given and retained and the 4-hour urine specimen compared in volume with that obtained when the test was made on admission 10 days previously. A bilateral subtotal thyroidectomy was done. Twelve hours after operation the patient became very restless, the pulse accelerated from 120 to 160 per minute, and in spite of continuous oxygen and intravenous glucose and iodine, hyperpyrexia to 105 degrees and delirium soon followed. Ice packs reduced body temperature to 102 degrees but the rapid, violent heart pounding persisted and the patient expired 22 hours after operation in typical thyroid crisis. Permission for autopsy was denied.

This was the only postoperative death in this series, and is the first postoperative fatality from any cause in 370 consecutive thyroidectomies.

The other patient (38, Table III) a woman 47 years of age, had a 19 year old toxic nodular goiter

complicated with auricular fibrillation and moderate congestive heart failure. There had been a weight loss of 25 pounds in the past year, 15 pounds of which were lost during the month prior to admission. After 16 days of routine treatment plus digitalis, a hemithyroidectomy was done. In this instance the results of the preoperative liver function test (25 per cent) were not known until after the patient had been operated upon. Sixteen hours after operation body temperature reached 102.4 degrees where it was controlled with cold sponges. The pulse was rapid, thready, and irregular, and the patient comatose. An extremely stormy postoperative course followed during the next 4 days. Despite full dosage of digitalis and continuous oxygen, cyanosis and dyspnea were prominent. A fasting blood sugar of 356 milligrams per cent was obtained 30 hours after operation. Small infusions of hypertonic glucose and insulin sufficient to maintain normal blood sugar values were given frequently. The 2 day postoperative liver function test was omitted because of the patient's condition; however, clinical jaundice was observed on the evening of the second and on the third postoperative days. Specific measures in addition to glucose and insulin were instituted as soon as nourishment could be taken orally. On the eleventh day after operation, 2 days before dismissal, the hippuric acid test had risen to 63 per cent of normal. Several months later liver function was 90 per cent, and the second stage of the operation was done with essentially no postoperative reaction and an uneventful convalescence.

Five of the patients in the "treated" group (Table III, patients 4, 22, 87, 89, and 119) appeared far more toxic on admission than either of the 2 patients referred to. One entered the hospital in crisis, one developed a beginning crisis 2 days after admission and a third had a toxic psychosis. All responded to specific therapeutic measures and, following surgery, no treatment other than routine was required except in the patient who developed lobar pneumonia on the first postoperative day (Table III, No. 87).

EFFECT OF THYROIDECTOMY ON LIVER FUNCTION

Nearly all patients subjected to thyroidectomy under local anesthesia for removal of a toxic goiter show a drop of 10 to 20 per cent in liver function on the second postoperative day (Fig. 2). This is transient. By the end of a week, liver function has returned to its preoperative level in nearly all instances. This impairment of liver function as a result of surgery is not due to the preoperative sedation and the local anesthetic (2 ounces of 0.5 per cent novocain). Patients with nontoxic nodular goiter or nontoxic fetal adenoma do not show this drop in liver function following thyroidectomy (Table IV.) Likewise, other patients subjected to surgery other than thyroidectomy (glands or tumors in the neck, benign breast tumors) showed no postoperative impaired liver function following the administration of the same preoperative sedation and novocain given the goiter patients. The damaging effect of thyroidectomy on liver function must therefore be due to some interrelationship between a *toxic* goiter and the liver. The exact nature of this influence is not known at present.

The comparative data in Table IV is significant. Here it is seen that the average drop in postoperative liver function in a group of patients subjected to hemithyroidectomy was 10 per cent as compared to a drop of 23 per cent in patients undergoing bilateral thyroidectomy. The two stage operation was selected for the former because they were obviously poorer operative risks than the latter. It does not seem that this observed difference in postoperative reaction can be explained satisfactorily on the basis of additional surgical trauma inflicted when both lobes, instead of one, are removed. Only several minutes are required to remove the second lobe after the first is excised. The preoperative medication and quantity of anesthetic are the same for both groups. Equally untenable is the idea that postoperative reaction following thyroidectomy results from thyroid secretion "squeezed" into circulation during the operative manipulation of the gland. Yet it is a widely observed fact that the 2 stage operation may mean the difference between a fatal postoperative reaction and uneventful recovery. Our data on liver function support the following explanation: In toxic goiter an excess of an abnormal thyroid secretion is poured into circulation, causing excessive stimulation of metabolic processes, particularly in the liver. It seems likely that in severe, untreated thyrotoxic patients the liver has been stimulated to a point where its stored principles are depleted and the majority of hepatic functions are depressed. Corrective therapy, directed at improving specific liver functions, overcomes or

TABLE IV.—EFFECT OF HEMITHYROIDECTOMY AND BILATERAL THYROIDECTOMY ON LIVER FUNCTION

Patient age	Clinical diagnosis	Pre-operative Treatment	Days	Liver function Per cent pre-operative	Per cent 2 days post-operative	Per cent difference
Nontoxic nodular and fetal adenoma (not degenerated) bilateral thyroidectomy						
9 45	Fetal adenoma	Routine	3	84	79	
16 24	Nontoxic nodular	Routine	4	92	90	
30 46	Nontoxic nodular	Routine	5	100	100	
148 31	Nontoxic nodular	Routine	4	100	96	
149 46	Fetal adenoma	Routine	3	100	98	
		Average		95	92	3
Toxic nodular and diffuse. Bilateral thyroidectomy						
4 42	Exophthalmic	Glucose-insulin	3	94	66	
5 37	Toxic nodular	Routine	8	85	65	
21 38	Toxic nodular	Routine	10	98	73	
52 45	Diffuse toxic	Liver extract, bilron	7	85	65	
63 49	Diffuse toxic	Liver extract, bilron	3	95	82	
90 27	Diffuse toxic	Liver extract, bilron	3	92	73	
91 38	Diffuse toxic	Liver extract, bilron	3	100	84	
113 18	Toxic nodular	Routine	8	96	53	
127 26	Toxic nodular	Routine	5	92	70	
54 34	Exophthalmic	Works	9	92	67	
		Average		93	70	23
Hemithyroidectomy. Poor risk patients						
10 37	Exophthalmic	Routine	10	52	51	
21 62	Degen. colloid	Routine	6	85	56	
71 49	Diffuse toxic	Works	8	92	83	
89 72	Diffuse toxic	Works	35	80	78	
119 56	Diffuse toxic	Works	19	81	77	
131 37	Diffuse toxic	Works	11	73	62	
171 52	Diffuse toxic	Works	8	85	70	
		Average		78	68	10

compensates in part the excessive demands made on the liver in thyrotoxicosis. Removal of both lobes of a toxic goiter produces a sudden withdrawal of the stimulating thyroid factor which has been present in excess. In the absence of overstimulation there follows a period of inertia during which the liver performs its functions sluggishly. In contrast, gradual withdrawal of the secretion of a toxic goiter, as in hemithyroidectomy, permits the liver to become readjusted by degrees. Whatever the answer, we do not subscribe to the prevailing theory that surgical trauma alone is the reason why "stage" operations have lowered surgical mortality and postoperative reaction in severely toxic goiters. This question deserves further study.

We have reported elsewhere studies on the effect of different anesthetics on liver function following various surgical procedures (33). Ether and ethylene have been found to result in diminished liver function varying from 20 to 50 per cent. Spinal anesthesia, in the absence of a fall in blood pressure, and local anesthesia (novocain) produce no demonstrable alteration of liver function in general surgical patients. If impaired liver function is to be minimized following thyroidectomy, local anesthesia is by far the anesthetic of choice. In this clinic no other anesthetic has ever been employed for thyroidectomy except in children.

RATIONALE OF CORRECTIVE HEPATIC THERAPY

Morphologically two types of cells in the adult liver are responsible for the many and varied hepatic functions. The reticulo-endothelial system (Kupffer cells), lining the hepatic sinusoids, serves to clear hepatic blood of particulate matter—bilirubin, foreign material, bacteria, possibly certain excretory products. Formation of antibodies and antitoxins also is thought to occur in the Kupffer cells. Metabolic, synthetic, detoxicatory, and storage functions are all carried out in the polygonal cells of the hepatic epithelium. In the liver there is no differentiation of special cells to perform special functions. Each hepatic cell is a unit in itself, contributing its share to the multifold functions attributed to the liver as an organ.

Much has been written about the liver's enormous functional reserve and its ability to regenerate, two remarkable characteristics of one of the largest and most vital organs. Experimental studies on partially hepatectomized animals, deprived of as much as 75 per cent of their liver without evidence of hepatic insufficiency, have been cited again and again

to substantiate the view that significant hepatic insufficiency is unlikely unless extensive liver damage has been inflicted. Is such a conclusion warranted? There is an infinite difference between the functional capacity of a liver retaining unimpaired function in a sizable portion of its substance and one in which every single hepatic cell is compromised. It is well known clinically that diseases of the liver characterized by "patchy" destruction of parenchyma—cirrhosis, metastatic new growths, cyst, or abscess—evidence liver insufficiency late in their course. In contrast, diffuse injury afflicting all cells alike—chloroform, phosphorous or arsenic poisoning, yellow atrophy—are characterized by early hepatic insufficiency. In toxic goiter we are dealing with a process that affects every cell in the hepatic parenchyma. If one important hepatic function, such as that of detoxication which involves both synthesis and conjugation, is impaired, it seems likely that various other functions performed by the same cell must also be affected, though not necessarily in like degree. We have seen repeatedly abnormal glucose tolerance curves and subnormal prothrombin values in toxic goiter patients with low hippuric acid excretion. All three return to normal after thyroidectomy and can usually be raised to normal before operation by specific corrective measures. The object of specific therapeutic measures is improved liver function, so that the patient is made the best possible operative risk. Each of the measures used in this study has a rational basis and is directed at improving specific liver functions or supplying materials that have been depleted or diminished in an overtaxed liver.

Glucose. Glucose offers, perhaps, the most valuable single means of improving liver damage. The ability of glucose to protect against liver damage is well established. It is stored in the liver in the form of glycogen. A liver high in glycogen withstands damaging procedures far better than one depleted of glycogen. Furthermore, glycogen depletion is associated with fatty infiltration of the hepatic cells. Fatty livers are more susceptible to damage than normal livers. The first consideration in treatment then is to load the liver with glycogen. A high carbohydrate diet with intermittent feedings of candy, fruit juices sweetened with dextrose, and fresh fruits, can yield up to 600 grams of glucose daily. This may be augmented in the poor risk patient or one with poor appetite and inadequate caloric intake by parenteral administration of glucose in water. Daily infusions of 1000 cubic centimeters of the 10 per cent solution or from 1 to 2 liters of the isotonic 5 per cent solution are ordinarily well tolerated. For patients with cardiovascular disease 300 to 500 cubic centimeters of 20 per cent glucose given slowly intravenously are of value a day or two preceding operation. If such hypertonic solutions are given rapidly one not only risks overloading the cardiovascular system but may induce a diuresis that could dehydrate the patient if pushed to the limit. This is decidedly undesirable in a prospective candidate for surgery.

Insulin. As a result of critical observation, we believe that conservative insulin therapy has a place in the management of severely toxic goiter. Many will disagree with this view. The literature abounds in reports to the effect that insulin, administered to normal laboratory animals, produces a transient increase in liver glycogen at the expense of blood sugar. The resulting hypoglycemia supposedly then accelerates the breakdown of hepatic glycogen to glucose. The end-result is thus diminished rather than increased liver glycogen. We do not doubt that insulin may produce such effects in normal animals and humans. However, a patient with a toxic goiter is not normal. A tendency to postcibal hyperglycemia and glucosuria is widely recognized in toxic goiter. There is good evidence that the removal from the portal blood and storage of glucose in the liver as glycogen is impaired (10, 20, 31). At the same time, increased demand for, and oxidation of, glucose by the tissues is a prominent feature of thyrotoxicosis, and in severe states of the disease the liver may be depleted of glycogen. These features suggest a refractiveness of the thyrotoxic patient to insulin. On the other hand, hypersensitivity to adrenalin is a characteristic of toxic goiter. It is thought that excessive liberation of thyroxin, or a precursor substance arising from a toxic goiter, either stimulates excessive adrenalin secretion or sensitizes

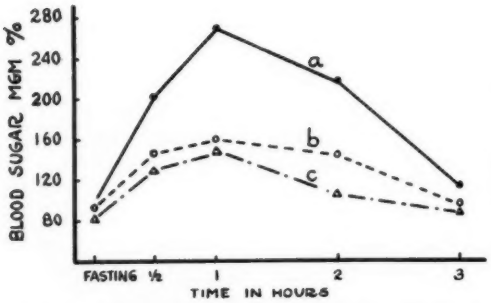

Fig. 3. Glucose tolerance in toxic goiter. Effect of bilateral thyroidectomy performed in 12 patients: *a*, tolerance curve before operation; *b*, 5 days after operation, *c*, normal curve.

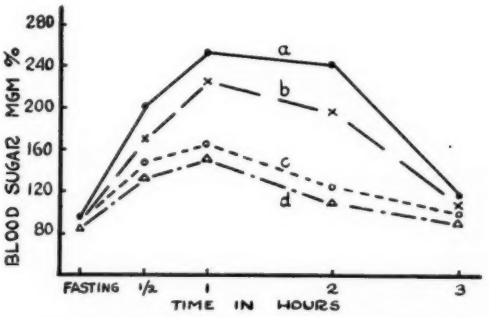

Fig. 4. Glucose tolerance in toxic goiter. Effect of hemithyroidectomy in 5 patients: *a*, before operation, *b*, after hemithyroidectomy, *c*, after removal of second lobe several months later, *d*, normal curve.

sympathetic nerve endings in the liver and tissues so that they respond overactively to normal amounts of adrenalin. Adrenalin induces glycogen depletion by accelerating the breakdown of muscle glycogen to lactic acid and of hepatic glycogen to glucose (9). Insulin has an inhibitory effect on adrenalin glycogenolysis (19). A contributing factor to the hyperglycemia of thyrotoxicosis may be increased hepatic formation of glucose from noncarbohydrate sources (gluconeogenesis). Insulin depresses gluconeogenesis.

While the thyrotoxic patient may not have an absolute deficiency of insulin, at least following the ingestion of carbohydrate there is for several hours a *relative* insulin deficiency. This is well demonstrated in the glucose tolerance curves shown in Figure 3. In these observations blood sugar specimens were obtained in the fasting state and at ½, 1, 2, and 3 hours following the ingestion of 1.75 grams of dextrose per kilogram of body weight. Capillary (arterial) blood was used to eliminate the factor of glucose utilization in the tissues. With this technique the maximum height of blood sugar rise, reached at the end of 1 hour, does not normally exceed 160 milligrams per cent. In a few of these patients glucose tolerance curves were obtained following the intravenous injection of the same quantity of dextrose given orally. Two hours were required for the injection. In general, the same type of glucose tolerance curve as that following the oral test was obtained, i.e. an abnormally high peak maintained over a prolonged period. This indicates that the observed high blood sugar values obtained at ½, 1, and 2 hours (Figs. 3 and 4) are not due to accelerated absorption of glucose from the alimentary tract, a theory held by some investigators (1). All of the patients represented in Figure 3 spilled sugar in the urine in the preoperative test. Removal of the goiter was followed by an immediate return to essentially normal glucose tolerance without glucosuria when the test was repeated 5 days after thyroidectomy (Fig. 3). When only half of the goiter was removed the disturbance in glucose tolerance was ameliorated but not corrected (Fig. 4).

In contrast to the abnormal glucose tolerance observed in toxic goiter, a number of tests on patients with nontoxic nodular goiters or fetal adenomas revealed normal glucose tolerance before operation. Removal of the goiter in these patients produced no effect on postoperative glucose tolerance, a fact confirming Gilligan's findings.

Wilder and Sansum found that normal subjects utilized intravenously administered glucose at a rate of 0.85 grams per kilogram per hour whereas thyrotoxic patients could handle only 0.50 to 0.65 grams per kilogram per hour without glucosuria. We found that 5 toxic goiter patients given 100 grams of glucose intravenously during a 2 hour period spilled from 10 to 20 grams of glucose in the urine. Addition of 20 units of regular insulin to the glucose solution eliminated the glucosuria.

Whether the retained glucose was converted to hepatic or muscle glycogen or was burned in the tissues is immaterial from a practical viewpoint. Presumably it was utilized. Improved liver function in one group of patients was obtained when insulin (5 units) was given by injection 3 times daily before meals. In these patients increased appetite and a larger caloric intake may have played a rôle in the improvement. It should be emphasized that insulin therapy is never used without assurance that a constant supply of glucose is available, either orally or parenterally. A detailed study of the effect of insulin on glucose tolerance in thyrotoxicosis is in progress and will be reported elsewhere.

Bile salts. Bile salts are manufactured by the hepatic cells exclusively. Animals deprived of bile by external fistula develop extreme weight loss, frequently show changes in behavior, and develop subcutaneous hemorrhages and bleeding from mucosal surfaces. The bone marrow becomes hyperplastic and the bony cortex decalcified. Frequently chronic duodenal ulcers are found at necropsy. Death occurs within several months. When all of the bile secreted is returned to the intestine of such animals a striking economy toward bile salts is shown. Approximately 90 per cent of the total bile salts are absorbed and retained in the enterohepatic circulation. The remaining 10 per cent is lost, but the liver continues to manufacture bile salts at a rate offsetting that lost each day. Thus a balance is kept, preventing a bile salt deficiency (32).

Experiments on animals or patients with biliary fistulas have shown that the synthesis of bile salts is immediately suppressed by minor degrees of liver damage not demonstrable by the usual liver function tests (6, 15, 22, 23, 36). It seems likely that in the presence of measurable liver damage the manufacture of bile salts must be diminished significantly. Prolonged impaired synthesis could result in a relative bile salt deficiency since the liver would be unable to replace the bile salts normally lost each day. We consider all toxic goiter patients with liver function under 75 per cent of normal in this category.

Bile salts are essential for the maximum digestion and absorption of foodstuffs, including the fat soluble vitamins A, D, E, and K as well as the minerals calcium and iron. From a practical point of view other effects worthy of consideration have been described. Administration of cholic acid to fed and fasting animals has been reported to increase liver glycogen significantly in acute experiments (24, 39, 40), however, there is no evidence that feeding normal animals bile salts over prolonged periods results in increased glycogen storage in the liver. On the contrary, Ivy (4) has recently observed that prolonged feeding of oxidized bile salt preparations to dogs and rats was without effect on liver glycogen. Intravenous injection of a purified bile salt derivative (decholin) has been reported to double the flow of arterial blood through the hepatic artery without affecting the rate of flow or pressure in the portal system (34). Orally administered oxidized bile salt preparations (decholin, ketochol, and kebilac) have also been found to increase hepatic arterial blood flow (16). This is an important consideration in regard to oxygenation of the liver cells, inasmuch as approximately three-fourths of the blood reaching the liver is venous blood from the portal vein. Increased arterial blood flow without a corresponding increase in portal flow would bring more oxygen to the hepatic parenchyma.

From results obtained clinically in toxic goiter and other states of hepatic insufficiency, we believe that the administration of bile salts is indicated in the treatment of hepatic insufficiency regardless of its cause. In instances in which oral administration is impractical we have used a purified bile salt derivative, sodium dehydrocholate (decholin), with glucose intravenously with results more striking than when glucose alone was given. Such therapy is of course contraindicated in the presence of obstruction to the common bile duct or acute, widespread degeneration of the hepatic parenchyma.

Liver principles. The normal liver stores certain essential substances other than glycogen where they can be withdrawn in times of emergency. Examples are antianemic principles and vitamins, particularly vitamin A and those of the B complex. In a damaged liver the function of storage is diminished and

reserves are limited. The increased metabolic turnover, characteristic of toxic goiter, must be met by increased supply of calories and essential materials lest a deficiency arise. Furthermore, the use of a high carbohydrate diet increases the requirement for certain constituents of the vitamin B complex (thiamin and riboflavin) which are essential to intermediary oxidation of carbohydrate and to oxidative reactions in the cells of all tissues. Some of the symptoms of thyrotoxicosis could be explained on the basis of vitamin B deficiency. There is good experimental evidence that large doses of thyroid extract deplete the liver of vitamin B and, conversely, that vitamin B therapy protects the liver against damage when the same amount of extract is fed (12, 13, 26, 38). It is assumed that the condition which results from excessive thyroid extract feeding to animals and thyrotoxicosis in humans are comparable states. Such an assumption is open to question and cannot be taken too literally.

In addition to administration of vitamin B complex, we have used a concentrate of liver (Wilson) representing 100 grams of fresh liver per gram of extract. This preparation is rich in substances normally stored in the liver, known and unknown, including vitamin B fractions. In addition, liver concentrate contains large amounts of choline, a substance which appears to play a rôle in the mobilization and metabolism of liver fat. Experimental studies are in accord that depletion of liver glycogen is accompanied by deposition of intracellular liver fat. Degenerative changes in the liver cells are always preceded by fatty metamorphosis unless the causative agent is overwhelmingly toxic. Fatty livers of animals can be prevented and cured by the feeding of choline (5, 17, 25), and we have observed patients with liver damage who responded slowly to glucose therapy alone markedly benefited by choline fed in the form of pancreatin or liver extract. Preliminary observations indicate that parenterally administered liver extract may be of comparable value in the treatment of hepatic insufficiency when oral therapy is impractical.

Glycine. Thyrotoxicosis is characterized by increased breakdown of body protein, a negative nitrogen balance, excessive destruction of phosphocreatin with creatinuria, and reduced synthesis of glycine by the liver cells. The feeding of glycine aids in sparing body protein, furnishes glycine for resynthesis of muscle phosphocreatin an important constituent necessary for release of energy in muscular contraction, and makes available a substance that can be used by the liver for conjugation and detoxication.

SUMMARY AND CONCLUSIONS

Of 207 consecutive goiter patients subjected to thyroidectomy 113, 55 per cent, showed evidence of hepatic insufficiency as measured by the Quick hippuric acid test. In 34 patients, 17 per cent, hippuric acid excretion was less than 70 per cent of normal, indicating appreciable impairment of liver function. Hepatic insufficiency appeared more related to the duration and intensity of the thyrotoxic state rather than to the type of goiter.

No consistent correlation between impaired liver function and basal metabolic rate was observed. Most extensive impairment of liver function was encountered in patients with goiters of long duration having normal or slightly elevated basal rates, and in thyroid crisis. Thyroid crisis and toxic psychosis were preceded or accompanied by evidence of marked hepatic insufficiency.

Serial determinations of liver function were made on toxic goiter patients during preparation for surgery and following operation. In this manner quantitative data were obtained to evaluate the therapeutic measures used during the preoperative period. The hepatic insufficiency present in toxic goiter is not permanent and can be greatly improved by specific measures directed at specific hepatic functions. The rationale of specific corrective hepatic therapy is discussed. Patients receiving this form of treatment showed greater improvement and were better operative risks than the control group of patients treated only by conventional measures now in general use.

The effect of thyroidectomy on glucose tolerance in toxic goiter was studied. The disturbance of carbohydrate metabolism as reflected by glucose tolerance is restored to essentially normal within 5 days after bilateral

thyroidectomy. Hemithyroidectomy ameliorates but does not correct the diminished glucose tolerance of thyrotoxicosis.

Thyroidectomy in toxic goiter has a damaging effect on liver function. This is not due to preoperative sedation and the local anesthetic used, because a number of nontoxic goiter patients and other surgical patients receiving the same medication failed to show postoperative evidence of liver damage. Local anesthesia is considered the anesthetic of choice for thyroidectomy.

It is believed that postoperative morbidity and mortality in severely toxic goiter can be decreased significantly by the use of specific therapeutic measures directed at improving liver function. We have had only the 1 postoperative death described in the text following 370 consecutive goiter operations. This is a mortality rate of 0.27 per cent.

REFERENCES

1. ALTHAUSEN, T. L. J. Am. M. Ass., 1940, 115:101.
2. BARTELS, E. C. Ann. Int. Med., 1938, 12: 652.
3. BEAVER, D. C., and PEMBERTON, J. DEJ. Ann. Int. Med., 1933, 7: 687.
4. BERMAN, A. L., SNAPP, E., IVY, A. C. and ATKINSON, A. J. Am. J. Digest. Dis., 1940, 7: 280.
5. BEST, C. H., and RIDOUT, J. H. Annual Review of Biochemistry, Stanford University, 1939, 8: 349.
6. BOLLMAN, J. L., and MANN, F. C. Am. J. Physiol., 1936, 116: 214.
7. BOYCE, F. F., and MCFETRIDGE, E. M. Arch. Surg., 1938, 37: 427.
8. CAMERON, G. R., and KARUNARATNE, W. A. E. J. Path. & Bacteriol., 1935, 41: 267.
9. CORI, C. F. Physiol. Rev., 1931, 11: 143.
10. CRAMER, W., and KRAUSE, R. A. Series B. Proc. Roy. Soc., London, 1913, 86: 550.
11. DINSMORE, R. S. J. Am. M. Ass., 1937, 109: 179.
12. DRILL, V. A., and HAYS, H. W. Proc. Soc. Exper. Biol. & Med., 1940, 43: 450.
13. DRILL, V. A., and SHERWOOD, C. R. Am. J. Physiol., 1938, 124: 683.
14. GILLIGAN, D. R., ABRAMS, M. I., and STEARN, B. Am. J. M. Sc., 1934, 188: 790.
15. GRAY, H. K., BUTSCH, W. I., and MCGOWAN, J. M. Arch. Surg., 1938, 37: 609.
16. GRODINS, F. S., OSBORNE, S. L., IVY, A. C., and GOLDMAN, L. Am. J. Physiol. 1941, 132: 375.
17. GYORGY, P., and GOLDBLATT, H. J. Exper. Med., 1940, 72: 1.
18. HAINES, S. F., MAGATH, T. B., and POWER, H. M. Proc. Staff. Meet. Mayo Clin., 1939, 14: 495.
19. HEGNAUER, A. H., and CORI, G. T. J. Biol. Chem., 1934, 105: 691.
20. KURIYAMA, S. J. Biol. Chem., 1918, 33: 193.
21. LAHEY, F. H. New England J. Med., 1935, 213: 475.
22. MCGOWAN, J. M., BOLLMAN, J. L., and MANN, F. C. J. Pharmacol., & Exper. Therap., 1936, 58: 305.
23. MCMASTER, P. D., BROWN, G. O., and ROUS, P. J. Exper. Med., 1923, 37: 395.
24. MIKI, T. Biochem. Ztschr., 1932, 247: 445.
25. MONTGOMERY, M. L., ENTENMAN, C., and CHAIKOFF, I. L. J. Biol. Chem., 1939, 128: 387.
26. PETERS, R. A., and ROSSITER, R. J. Bio-Chem. J., 1939, 33: 1140.
27. PROBSTEIN, J. G., and LONDE, S. Ann. Surg., 1940, 111: 230.
28. QUICK, A. J. J. Biol. Chem., 1931, 92: 65.
29. Idem. Am. J. M. Sc., 1933, 185: 630.
30. ROWE, A. W. Endocrinology, 1928, 12: 1.
31. SANGER, B. J., and HUN, E. G. Arch. Int. Med., 1922, 30: 397.
32. SCHMIDT, C. R., BEAZELL, J. M., BERMAN, A. L., IVY, A. C., and ATKINSON, A. J. Am. J. Physiol., 1939, 126: 120.
33. SCHMIDT, C. R., UNRUH, R. T., and CHESKY, V. E. Effect of anesthetics and surgical procedures on liver function. In press.
34. SCHWIEGK, H. Arch. f. exper. Path. u. Pharmakol., 1932, 168: 693.
35. SHAFFER, J. M. Arch. Path., 1940, 29: 20.
36. SMYTH, F. S., and WHIPPLE, G. H. J. Biol. Chem., 1924, 59: 623.
37. SNELL, A. M., and PLUNKETT, J. E. Am. J. Digest Dis., 1936, 2: 716.
38. SURE, B., and SMITH, M. E. J. Nutrition, 1934, 7: 547.
39. TANAKA, K. J. Biochem., 1932, 14: 463.
40. WATANABE, K. Biochem. Ztschr., 1932, 255: 155.
41. WELLER, C. V. Ann. Int. Med., 1933, 7: 543.
42. WILDER, R. M., and SANSUM, W. D. Arch. Int. Med., 1917, 19: 311.
43. YOUMANS, J. B., and WAREFIELD, L. M. Arch Int. Med., 1926, 37: 1.

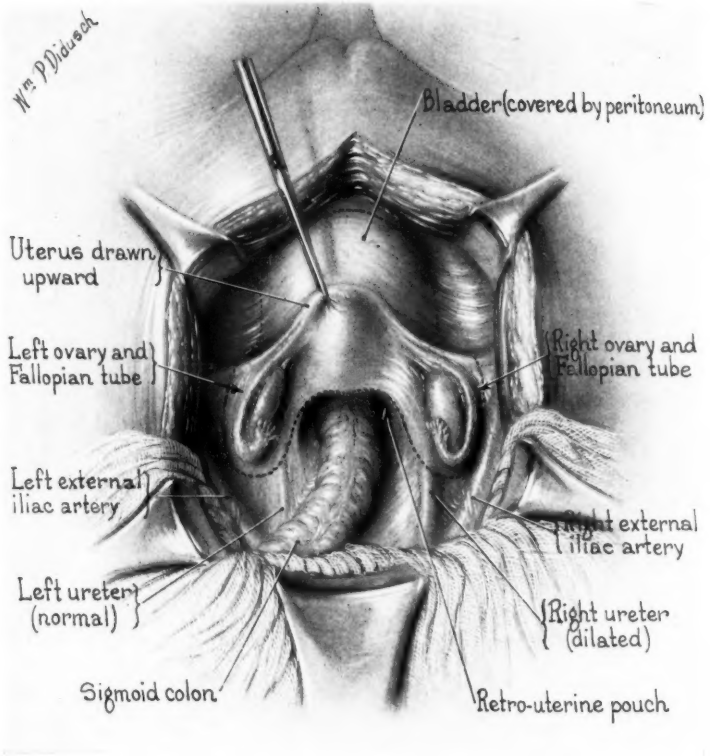

Fig. 1. Dotted line indicates extent of peritoneum removed.

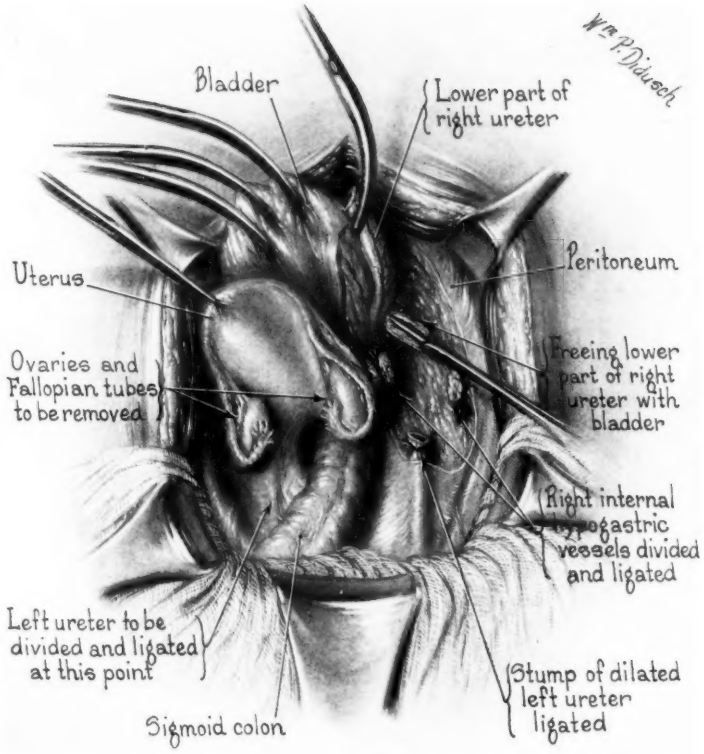
Wm. P. Didusch
Bladder
Lower part of right ureter
Uterus
Peritoneum
Ovaries and Fallopian tubes to be removed
Freeing lower part of right ureter with bladder
Right internal epigastric vessels divided and ligated
Left ureter to be divided and ligated at this point
Stump of dilated left ureter ligated
Sigmoid colon

Fig. 2. Further steps in operation.

Total Cystectomy Combined with Panhysterectomy.—Samuel A. Vest and Howard H. Curd

TOTAL CYSTECTOMY COMBINED WITH PANHYSTERECTOMY

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EXTENSIVE infiltrating carcinoma of the bladder is one of the most discouraging conditions encountered in urology. Radical surgery, although accompanied by relative high mortality, is often the only hope of cure. Only a few extensive infiltrating carcinomas have been reported to be radiosensitive with disappearance of the tumor following adequate deep x-ray therapy. Gross disappearance in most cases has been temporary with recurrence of the tumor later. The effect of deep x-ray, radium, and radon in extensive tumors is at best palliative (2, 3, 4) so that there is every justification for radical surgery whenever possible.

It is well known that extensive tumors of the bladder can exist for considerable periods of time, even years, before metastases occur; hence the bladder in some respects is a favorable site for the application of radical surgery. Unfortunately, a considerable majority of these cases are not suitable for surgery. A great many infiltrating carcinomas involve the base of the bladder, including the trigone and ureteral and vesical orifices, thus complicating the surgical problem.

Total removal of the bladder with diversion of the urine is often the sole hope of cure, or at least relief, from distressing symptoms. Diversion of the urinary stream itself may often be the major problem. Transplantation of the ureters into the sigmoid is perhaps the most ideal procedure, but it is accompanied by appreciable immediate and late mortality. When the trigone area is involved, there is often obstruction of the ureteral orifices resulting in various degrees of dilatation of the upper urinary tract. Patients with dilated ureters and impaired kidneys are poor candidates for ureterosigmoidostomy and when such impairment is present nephrostomy or ureterostomy are the remaining possible procedures. Following diversion of the urine by one of these procedures, complete removal of the urinary bladder is a sound and well established procedure. Hinman has recently reviewed the literature on total cystectomy for cancer and, in 254 cases, the mortality was 34.2 per cent. In the 75 cases reported since 1926 there were 19 operative deaths, a mortality of 25.3 per cent. Such a mortality seems high, but it must be recognized that such patients are doomed and in addition many suffer from intolerable bladder symptoms. From his personal experience Hinman reported 6 of 21 patients living and well after total cystectomy for carcinoma.

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It is the purpose of this communication to report an operative procedure which proved invaluable to the authors in a recent case. After preliminary diversion of the urine by means of nephrostomy, an attempt was made to remove the entire urinary bladder, according to the usual technique. In so doing it was found that the carcinoma from the base of the bladder had extended locally through the bladder wall and was beginning to involve the cervix and vaginal vault. The usual technique of total cystectomy was then necessarily modified and combined with panhysterectomy.

Mrs. H. V. S., a 48 year old multipara, was admitted to the Urology Service of the University of Virginia Hospital April 1, 1939 with a history of occasional hematuria since her first pregnancy, 28 years previously. One year before admission she had noticed a gradual increase of bladder irritability with a dull pain in her right flank. She came to the hospital, however, because of sudden severe pain in the left flank associated with chills and fever. For 2 months before admission there had been intermittent hematuria and she had lost about 30 pounds.

Examination revealed a pale, anemic woman who showed evidence of weight loss. Abdominal examination revealed a mass below the right costal margin which was thought to be the right kidney. On vaginal examination a distinct area of induration at the base of the bladder could be felt through the anterior wall of the vagina. The uterus was of normal size, in good position, and freely movable. Cystoscopy revealed an extensive, irregular, papillary tumor involving the entire base of the bladder. It completely obscured the trigone and ureteral orifices and extended upward on the posterior wall for a few centimeters. This tumor began just proximal to the vesical orifice and extended about halfway up on each lateral wall. It was impossible to catheterize the ureteral orifices. Intravenous pyelograms revealed no function in the right kidney after an hour and a half. On the left there was considerable dilatation of the ureter and marked hydronephrosis of the upper half of the kidney. The patient was obviously living upon one-half of the left kidney with little or no renal reserve. The blood urea, however, was normal. X-ray films of abdomen and chest revealed no evidence of metastasis. Hemoglobin was 75 per cent and red cell count was 4.6 million. A biopsy specimen from the tumor showed an epidermoid carcinoma which was estimated to be somewhere between grade II and III malignancy. It was obvious that electrofulguration, radon, and deep x-ray therapy

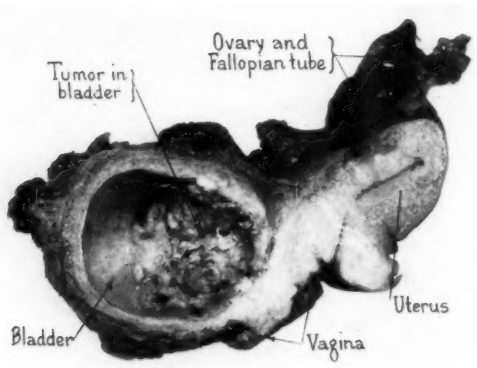

Fig. 3. Total specimen removed.

could offer nothing beyond mere palliation for such an extensive tumor.

A left nephrostomy was performed under spinal anesthesia to relieve the bladder symptoms. Following this the patient voided no urine, indicating that the right kidney was completely blocked. The intense bladder symptoms were entirely relieved and the general strength of the patient improved considerably. After much deliberation it was decided to carry out abdominal exploration with the hope that no tumor would be found outside the bladder and a total cystectomy could be performed.

Operation was performed on April 25, 1940. A low, midline, abdominal incision was made from the symphysis to the umbilicus. A careful search was made for evidence of metastases. No palpable lymph nodes were found anywhere in the pelvis or along the aorta. The liver was smooth. A large hydronephrotic, apparently functionless, kidney was present on the right. A large area of induration involving the base of the bladder could be palpated through the anterior bladder wall. The induration did not seem to extend laterally from the confines of the bladder itself. This induration seemed to involve the vaginal vault and cervix and it was obvious that there was no hope of curing the patient unless the cervix, uterus, and upper third of the vagina were removed with the bladder. As this presented the only possibility of cure, the following operative procedure was carried out.

The peritoneum was divided at its reflection from the bladder anteriorly as indicated by Figure 1. The dotted line indicates the extent of peritoneum removed with the operative specimen. The round ligaments were next divided and ligated on both sides. The lateral aspects of the broad ligaments, together with the ovarian arteries, were next divided and ligated. The ureters were then isolated near the brim of the pelvis, divided, and ligated as shown in Figure 2. The distal portions of the ureters, consisting of the last 3 inches, were then dissected up from the floor of the pelvis as shown in Figure 2 and carried forward with the base of the bladder and uterus. Because the ureters were carried forward as shown, the usual procedure of clamping and dividing the uterine arteries was not feasible. It was then decided to ligate the anterior branch of the internal iliac arteries on both sides. These were ligated, together with a moderate amount of surrounding tissue (Fig. 2), followed by division of the ureterosacral ligaments. Ligation of the anterior branch of the internal iliac made division of the ureterosacral ligaments fairly easy. The uterus was then completely free except for its vaginal and vesical attachments. It was easy to separate the bladder from the symphysis by blunt dissection and to isolate and divide the urethra. The lateral ligaments and blood vessels to the bladder were then clamped, divided, and ligated, so that the uterus and bladder were entirely free except for the vaginal attachments. The vagina was then freed from the rectum posteriorly, following which it was clamped and divided and the total specimen removed in one piece as shown in Figure 3.

A little over 2 hours were required to carry out the operative procedure described. At the end of this time the patient's blood pressure had begun to fall and it was necessary to give her intravenous fluids and a transfusion. It was deemed inadvisable to keep the patient on the operating table longer in order to attempt to reperitonealize the raw area in the pelvis and a sheet rubber dam was placed over the denuded area and brought out the lower angle of the wound. The peritoneal cavity was closed with a continuous suture of plain catgut and the rectus fascia closed with mattress sutures of No. 1 chromic catgut. The vagina was left open and a Penrose drain was inserted from above to the outside.

The patient did remarkably well following this radical procedure and in the subsequent days the pulse rate did not go above 100. There was a slight fever for the first 10 days. For the first few days after operation there was moderate postoperative distention following which there have been no gastrointestinal symptoms whatever. The rubber dam was removed from the lower end of the incision at the end of 1 week and after moderate drainage for a few days, healing promptly occurred. At the end of the second week a thrombophlebitis of the left leg developed with temperature going to 102.6 degrees. As a result of the phlebitis the patient was kept in bed for 3 weeks and at the end of 39 postoperative days she was ready to go home. It was decided to change the nephrostomy tube before discharge. This was unfortunately followed by a chill and a febrile course which delayed her final discharge for 12 additional days. She then left the hospital in good condition, comfortable, and with a hemoglobin the same as on admission.

The patient returned to the hospital 2 months later greatly improved. She had learned to take care of the nephrostomy tube and seemed to be contented. She could go anywhere she wished and had no complaints. The nephrostomy catheter was inadvertently removed and in an effort to insert a larger catheter a false pocket must have been made because catheters could not be reinserted into the kidney. It was necessary to readmit the patient and perform a ureterostomy. The nephrostomy tube was later reinserted so that optimum drainage would take place from her only functioning kidney. This enabled through-and-through irrigation of the kidney to be carried out. She received 12,150 r units of deep x-ray therapy to the lower abdomen in the hope that any remaining malignant cells in the perivesical lymphatics might be destroyed. This was given in divided dosages over a period of 52 days by Dr. Vincent W. Archer, roentgenologist to the University Hospital. The patient is now in good general condition and is able to travel and care for her ureterostomy and nephrostomy tubes. She is comfortable, has no complaints, and seems to be satisfied with the results. There have been no symptoms whatever from the nonfunctioning right kidney.

The pathological specimen, Figure 3, consisted of urinary bladder, uterus, tubes, ovaries, a portion of the vagina, the lower several inches of both ureters, broad and round ligaments. It can be seen that the entire floor and trigonal area of the bladder is the site of a raised, irregular, grayish mass, the diameter of which measures roughly 6 by 5 centi-

meters. The ureteral orifices could not be easily identified. The remainder of the bladder wall is normal. Infiltration of the bladder wall by the tumor is readily visible in the photograph and the point at which it was beginning to involve the vesicovaginal septum can be seen. Microscopic section revealed an infiltrating grade III epidermoid carcinoma.

It is obvious that a very radical, extensive procedure was carried out in the foregoing case. We feel that it was justified because any other form of therapy would have been only palliative. Radical surgery was the one chance of possible cure of this patient. It is entirely possible that a cure has not been achieved, and the final result may not be known for years. The patient has at least been entirely relieved of her distressing, almost intolerable, bladder symptoms for the 15 months which have elapsed since operation.

The purpose of reporting this case has been to call attention to the possibility of removing the entire urinary bladder and at the same time combine it with a panhysterectomy. The operation is made feasible by ligation of the anterior branch of the internal iliac artery below the brim of the pelvis. At the time this operation was performed we were unaware that the late Franklin H. Martin (6) first proposed such a procedure for either cancer of the uterus, which had extended to the bladder, or cancer of the bladder, which had extended to the uterus. He believed that it was possible to cure such cases in which all vestiges of malignant tumor could be removed. His technique of removal of the uterus and bladder was somewhat similar to the one which we have used except that he inserted clamps through the vagina for several days to hold the broad ligaments and uterine arteries. The clamps were removed later after obliteration of the vessels had occurred. The operation was carried out by Martin in 1899, at which time he transplanted the ureters into the rectum for diversion of the urine. The patient unfortunately died 4 hours later in shock.

In a review of the literature a similar case was reported by Walton Martin (7) in 1910. He brought the ureters out to the skin margin and stated that at the end of 35 days the patient seemed to be doing well. Antonucci also mentions carrying out a similar operation in 1928. In a very recent publication Vas Concellos and Da Costa report the use of an identical technique to remove the bladder and internal generative organs. They also stress the ligation of the internal iliac below the origin of the gluteal vessels in order to control bleeding.

The technical success in our case is indicative of the feasibility of carrying out such an extensive operation as total cystectomy combined with panhysterectomy. Such a procedure as we have reported is obviously indicated only in an occasional case. Although our patient is living and well only 15 months, this report will call attention to a valuable operation in the hope that more cases in which it is applicable will be recognized.

SUMMARY

1. Extensive carcinoma of the bladder is one of the most distressing and almost surely fatal conditions encountered in urology. Evidence indicates that fulguration, radon, and deep x-ray therapy at the present time offer only palliation.

2. Radical surgery is indicated in extensive carcinoma of the bladder when there is no clinical evidence of extension beyond the bladder wall.

3. A case is presented in which patient suffered from a large epidermoid carcinoma of the bladder infiltrating the base and involving the adjacent portion of the vagina but without evidence of metastasis or extension beyond this area.

4. A surgical technique is shown illustrating the feasibility of removing the entire urinary bladder, together with the uterus, broad and round ligaments, ovaries, tubes, cervix and upper portion of the vagina *en masse*. This procedure, although radical, offers the only hope of cure in such cases as reported here.

REFERENCES

1. Antonucci, C. J. de chir., 1928, 32: 153.
2. Ash, J. E. J. Urol., 1940, 44: 135.
3. Barringer, B. S. J. Urol., 1938, 40: 606.
4. Colby, F. H. J. Urol., 1940, 44: 216.
5. Hinman, Frank. Surgery, 1931, 6: 851.
6. Martin, Franklin H. Am. Gynæc. & Obst. J., 1900, 16: 395.
7. Martin, Walton. Ann. Surg., 1910, 51: 126.
8. Vas Concellos, E., and Da Costa, J. B. Ann. Paulistas de med. e cir., 1940, 39: 127.

THE END-RESULTS OF THE TREATMENT OF MALIGNANT TUMORS OF THE PALATE

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THE most common malignant tumors of the palate are squamous cell epitheliomas and adenocarcinomas of the mixed tumor type. In order to determine the results obtained in the treatment of malignant tumors of the palate at the Mayo Clinic, we have reviewed the histories and pathological specimens of 173 cases of malignant tumors in this region which were observed in the years 1907 to 1939, inclusive. During this same period 236 cases of benign tumors of the palate were observed. This makes a total of 409 cases of tumor of the palate. Table I shows the relative incidence of the various benign and malignant tumors of the palate and the sex of the patients.

ADENOCARCINOMA OF MIXED TUMOR TYPE

Adenocarcinoma, mixed tumor type, presents a characteristic appearance in the palate. It is a hard, rounded tumor with a smooth mucous membrane over the surface, and it is sometimes found on routine examination. The most frequent site of such tumors in the head and neck is the parotid region; but, they may occur in the submaxillary region, floor of mouth, palate, larynx, pharynx, nasopharynx, tongue, upper lip, cheek, and orbit.

They have been given various names by pathologists and show many variations in their histological structure. They have been named fibromyxo-endothelioma, endothelioma, adenocystendothelioma, cylindroma, and chondrocarcinoma, depending upon the predominating type of cell present. Broders expressed the opinion that these tumors are all adenocarcinomas of the mixed tumor type, and this term was used by one of us (New) and Childrey in a report in 1931.

From 1907 to 1939, inclusive, 76 cases of adenocarcinoma, mixed tumor type, of the palate were observed at the Mayo Clinic. Figure 1 shows the various sites of this neoplasm in the head and neck.

When this neoplasm involves the palate, it frequently causes few, if any, symptoms until it becomes rather large. It grows so slowly that the enlargement does not interfere with the function of the pharynx or palate. Mechanical interference with deglutition and swallowing may be the only symptom if the tumor fills the upper part of the mouth. The patient may notice that the upper denture does not fit, owing to the gradual enlargement of the tumor. Patients may complain of nasal obstruction as a result of extension of the neoplasm into the floor of the nose.

The average age of the patients in the 76 cases at the time of examination was 50 years for males and 42 years for females; the youngest patient was 12 years of age, and the oldest, 80 years. This type of tumor occurs most frequently in the fourth and fifth decades. Table II shows the duration of symptoms. In many of the cases the tumor had been present for many years before it caused the patient to consult a physician. The fact that the tumor had been present for 10 to 20 years in 14 cases is evidence of this.

The tumor may fill the mouth almost completely and the patient may accommodate himself to its presence. The largest tumor was 9 centimeters in diameter; it was lobulated, cystic, and filled the mouth completely. The smallest tumor was 5 millimeters in diameter. Tumors of this type may extend into the nose and antrum or nasopharynx. In 2 cases there was direct intracranial extension through the ethmoid and sphenoid bones.

The average duration of symptoms in the 76 cases was 4.75 years; the shortest duration was 1 month, and the longest, 30 years. In almost half of the cases the tumor was situated at the junction of the hard and soft palate. This might be of some significance since this is the main location for the openings of the ducts of the mucous glands. In 19 of the 76 cases the tumor involved the hard palate; in 16 cases it involved the soft palate. It is rare to see a mixed tumor in the midline (Fig. 2); a tumor was found here in only 4 of the cases.

Usually adenocarcinomas of the mixed tumor type are rather hard, smooth and sessile, and oval in shape (Fig. 3). At times there are soft regions in the tumor, and some tumors are nodular. They are encapsulated to begin with (Figs. 4a and b) and frequently produce a saucer-shaped cavity in the bone of the hard palate. Ulceration was present on the surface in 15 of the cases.

From the Section on Laryngology, Oral and Plastic Surgery, Mayo Clinic and Mayo Foundation.

TABLE I.—TUMORS OF PALATE: INCIDENCE ACCORDING TO TYPE OF TUMOR AND SEX OF PATIENT

Type of tumor		Cases	Sex	
			Male	Female
Benign	Papilloma	125	101	24
	Adenoma	8	3	5
	Fibroma	16	8	8
	Hemangioma	8	6	2
	Lymphangioma	3	3	
	Lipoma	1		1
	Exostosis (torus palatinus)	74	24	50
	Foreign body giant cell tumor	1	1	
	Total for benign tumors	236	146	90
Malignant	Squamous cell epithelioma	84	75	9
	Adenocarcinoma (mixed tumor type)	76	36	40
	Melano-epithelioma	3	2	1
	Fibrosarcoma	4	1	3
	Fibromyxosarcoma	1	1	
	Myxosarcoma	1		1
	Chondrosarcoma	1		1
	Lymphosarcoma	3	2	1
	Total for malignant tumors	173	117	56
Total for benign and malignant tumors		409	263	146

The majority of the tumors contain regions of epithelial cells which fade into masses of mucoid material, connective or cartilaginous tissue. It is well known that the epithelial cells of the so called mixed tumors often show a great tendency to polymorphism. This is exemplified by the fact that in these tumors one finds oval cells, round cells, and spindle cells of various sizes. Columnar cells, cuboidal cells, and sometimes squamous cells with intercellular bridges or spines also are found. In the regions of squamous cells one occasionally finds pearly bodies.

The epithelial cells may be arranged in acini, tubules, or alveoli. The tubules often produce a vascular effect. The epithelial cells may also be arranged in solid masses or narrow strands of small, flattened, compact epithelium of basal type, which mingle with stellate, mucous, connective tissue or cartilage cells. Even in cases in which mucous connective tissue and cartilage are absent, the tumors can be recognized as adenocarcinomas of the mixed tumor type. The polymorphism and arrangement of the epithelial cells are the distinctive features. The neoplasm can be graded depending upon the appearance of the cell and the amount of mitotic figures present.

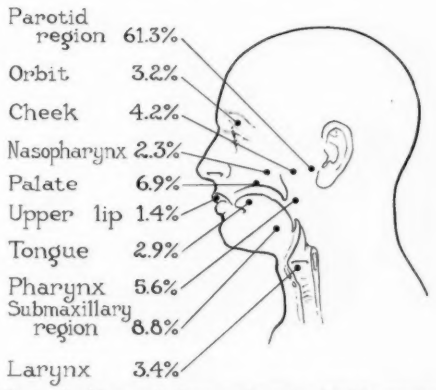

Fig. 1. Incidence of adenocarcinoma, mixed tumor type, of the head and neck according to the site of the lesion.

There should be little difficulty in diagnosing this type of tumor on the basis of the situation, the long duration, the movable, hard, nodular tumor. Fibromas or fibrosarcomas are about the only tumors that simulate adenocarcinoma of the mixed tumor type. Frozen sections are always prepared at the time of the operation and are examined microscopically.

Treatment. Adenocarcinomas of the mixed tumor type are removed surgically when it is at all possible. At times, preliminary ligation of the external carotid is performed, and an incision is made in the palate down to the capsule of the tumor. The tumor is shelled out by blunt or finger dissection. Nitrous oxide-oxygen and ether may be administered intratracheally or pentothal sodium may be given intravenously to produce an-

TABLE II.—DURATION OF SYMPTOMS IN ADENOCARCINOMA OF THE MIXED TUMOR TYPE

Duration of symptoms years	Cases	
	Number	Per cent
Less than 1	17	22.4
1 to 2	10	13.2
2 to 5	20	26.3
5 to 10	5	6.6
10 to 20	14	18.4
20 to 30	2	2.6
Lesion discovered accidentally	6	7.9
Duration not stated	2	2.6
Total	76	100.0

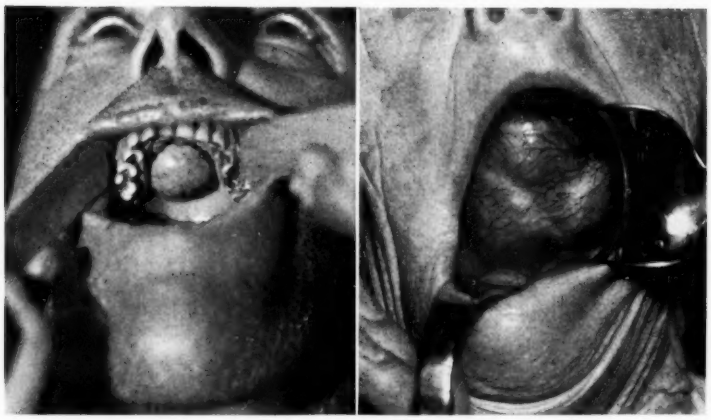

Fig. 2, left. Adenocarcinoma, mixed tumor type, in midline of palate.
Fig. 3. Adenocarcinoma, mixed tumor type, of the left side of the palate.

esthesia. If the tumor is attached to the periosteum of the bones of the palate, the bone is cauterized with surgical diathermy. If the tumor has extended up into the nose or maxillary sinus, a very wide removal with surgical diathermy is necessary. Radium seeds or needles are used at the time of operation if it is questionable whether the entire tumor has been removed. Perforation of the palate or of the nasal fossa sometimes results. This is later closed with a dental plate. If the tumor is so extensive that complete removal seems impossible, radium seeds or needles are inserted into the neoplasm, sometimes at repeated stages a few months apart. Although this type of tumor is generally not radiosensitive, much may be accomplished with this type of treatment, particularly in cases in which the tumor recurs.

In all but 1 of the 76 cases the tumor was removed through the mouth. In this case the patient had a large mixed tumor of the palate and maxillary sinus, and resection of the superior maxilla was performed in 1913. He has had several local recurrences since then, but he is well at the present time, 27 years after first operation.

In the 76 cases, the lymph nodes of the neck were not removed unless metastasis was present. Few of these tumors metastasize. In 13 of the cases the lymph nodes of the neck were palpable at the time of the first examination. In 4 of the 13 cases the enlarged lymph nodes were removed.

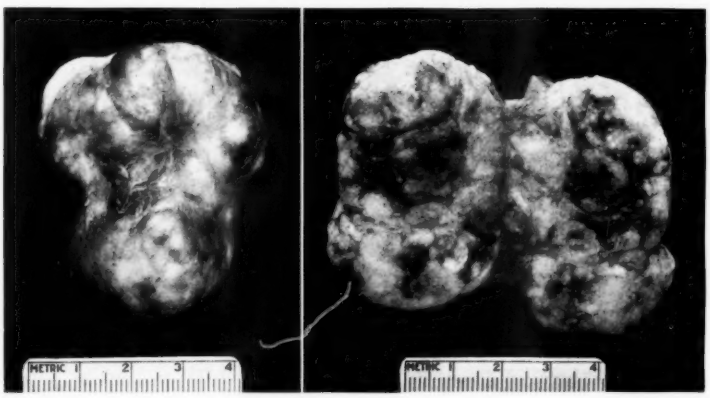

Fig. 4. a, left, Adenocarcinoma of mixed tumor type removed at operation. b, Gross appearance of cut section of lesion.

TABLE III.—FIVE YEAR AND TEN YEAR SURVIVAL RATES IN CASES OF ADENOCARCINOMA (MIXED TUMOR TYPE) OF THE PALATE

Treatment	Cases*	Patients traced*	Lived 5 or more years after treatment		Cases*	Patients traced*	Lived 10 or more years after treatment	
			Number	Per cent of traced patients*			Number	Per cent of traced patients*
Irradiation only (palliative)	7	7	3	43	5	4	1	25
Surgical diathermy and irradiation	14	14	13	93	10	10	9	90
Excision and surgical diathermy (with or without irradiation)	23	22	21	95	19	17	13	76
Excision (only)	9	9	9	100	8	6	6	100
Total	53	52	46	88	42	37	29	78

*Inquiry as of January 1, 1940. The 5 year survival rate was determined by a study of cases in which the patients were treated 5 or more years before this investigation, that is, in 1934 or earlier. The 10 year survival rate was determined by a study of cases in which the patients were treated in 1929 or earlier.

Microscopic examination of these revealed adenocarcinoma, grade 2 of the mixed tumor type.

In 25 per cent of the 76 cases operation, irradiation, or both had been employed before the patients came to the clinic. In 7 cases in which irradiation had been used alone before the patients came to the clinic, the tumors were so situated and fixed that they were considered inoperable; therefore, irradiation only was used at the clinic.

Results. The results of the treatment are shown in Table III. In 9 cases in which there was no recurrence following excision the tumors, which were encapsulated, were shelled out without rupture. In 23 cases in which surgical diathermy was used as well as excision the bone was involved, and it was necessary to destroy the periosteum or part of the bones of the palate or superior maxilla in order to remove the tumor. In the 14 cases in which surgical diathermy and irradiation were used the bone also was involved, and irradiation was used in addition to diathermy.

In the 4 cases in which enlarged lymph nodes in the neck were removed the duration of life was as follows: 1 year in 1 case, 4 years in 1 case, 9 years in 1 case, and in the remaining case the patient is still alive 32 years after operation. The large number of patients who lived 5 and 10 years after treatment tends to prove that most tumors of this type have a low grade of malignancy.

SQUAMOUS CELL EPITHELIOMA OF THE PALATE

Primary squamous cell epitheliomas of the palate are infrequent. Because of the fact that in a case of very extensive involvement it is difficult to tell just where the tumor originated, only those cases in which we believe that the tumor started in the palate are included in this series. Tumors that secondarily involve the palate, that is, those that started in the gum, tonsil, pharynx, maxillary sinus, or cheeks, were not included in this group. From 1907 to 1939, inclusive, 84 cases of primary squamous cell epithelioma of the palate were observed at the clinic. During this time approximately 5,000 cases of malignant tumors of the buccal cavity were observed.

Fig. 5. Squamous cell epithelioma, grade 2, of the soft palate.

Forty per cent of the 84 patients complained of soreness of the mouth. Twelve patients had trouble wearing their dental plates as the plate either did not fit or hurt them. The first symptom in 2 cases was enlarged cervical lymph nodes. One patient complained of bleeding from the mouth. In some cases the growth was found accidentally.

The average age of the patients was 58.2 years, and only 10.7 per cent of the patients were women. The duration of the tumors varied from a few weeks to several years. Sometimes the presence of a thickened leucoplacia may have been noted for years, and then the growth became active and underwent ulceration.

In 50 per cent of the cases the tumor was found in the soft palate, and in 25 cases it was situated in the hard palate alone. In 5 cases the mass was situated in the uvula and the adjacent part of the soft palate; in 2 cases the tumor was situated on the upper surface of the soft palate. In 26, or 30.9 per cent, of the 84 cases the tumor had extended into the adjacent structures, that is, into the nose, nasopharynx, maxillary sinus, and pharynx.

The low grade tumors appear as grayish ulcerated lesions and sometimes are papillary. The more malignant lesions have soft, easily bleeding surfaces. Although the diagnosis in all cases must

TABLE IV.—SQUAMOUS CELL EPITHELIOMA OF THE PALATE: INCIDENCE ACCORDING TO GRADE OF MALIGNANCY

Grade	Cases	
	Number	Per cent
1	11	15.3
2	25	34.7
3	30	41.7
4	6	8.3
Total	72	100.0

be made microscopically by using fresh frozen sections at the time of operation, usually it can be made by the appearance of the lesion (Fig. 5).

Treatment. We believe that removing the local growth with surgical diathermy under anesthesia produced by the intratracheal administration of nitrous oxide-oxygen and ether or by the intravenous use of pentothal sodium offers the best chance of getting rid of the lesion. A large sequestrum usually follows the removal, and the greater part of the soft palate is lost. A dental plate is required to close the opening. Irradiation is administered directly into or over the involved region in cases in which the lesion is active.

Nine patients with palpable glands had gland dissection in addition to the treatment of the local lesion. They all showed involvement microscopically. Of these, 6 returned later to the clinic for examination, and they lived on an average of 5½ years after the initial treatment. Seventeen patients received radium, x-ray, or both. Five did not receive any treatment at the clinic, the lesion being too far advanced, but they had palliative x-ray at home. The grade of the tumor and the results are shown in Tables IV and V.

OTHER MALIGNANT TUMORS

The malignant tumors other than the adenocarcinoma and squamous cell epithelioma that were encountered in the 173 cases are listed in Table I. The treatment of these tumors depends upon the activity of the growth. If the tumors are encapsulated, surgical removal is advisable. The use of diathermy and radium depends upon the extent of the lesion. Irradiation is also used externally.

Of the 3 patients who had melanoepithelioma and were treated with diathermy and radium, 1 lived 4 years after treatment, and the others, 2 years after treatment. In 1 of them the lesion was inoperable and palliative irradiation was used.

In 4 cases the patients had a fibrosarcoma. In 3 of the cases the tumor was removed surgically and

TABLE V.—RELATION OF FIVE YEAR SURVIVAL RATE TO GRADE OF MALIGNANCY IN CASES OF PRIMARY SQUAMOUS CELL EPITHELIOMA OF THE PALATE

Grade of tumor, on basis of 1 to 4	Cases*	Patients traced*	Lived 5 or more years after treatment	
			Number	Per cent of traced patients
1	5	5	5	100
2	20	16	7	44
3	21	21	8	38
4	5	5		
Not stated†	9	5		
Total	60	52	20	38

*Inquiry as of January 1, 1940. This group includes only patients who were treated in 1934 or earlier.

†In these cases the patients were treated in the years 1910 to 1918, inclusive.

the bone destroyed with surgical diathermy. In addition, 1 patient received radium treatment. Of these 3 patients, 1 had no recurrence in 5 years; 1 had none in 10 years, and 1 had none in 20 years, respectively, after the initial treatment. The fourth patient had a very extensive involvement of the palate and a fibrosarcoma, grade 4, involving lymph nodes. Radium was used as a palliative measure, but patient died 1 month later.

The patient who had a fibromyxosarcoma also had extensive involvement of the soft palate and of the lymph nodes in both cervical regions. This patient, a boy, aged 13 years, was treated with radium and x-rays but died 2 years later.

The patient with myxosarcoma of the palate, a girl, aged 7 years, had had difficulty in swallowing for 3 weeks and swelling of lymph nodes of neck for 2 months. She died a month after treatment.

The patient with chondrosarcoma of the palate, a woman, aged 49 years, had had a tumor removed from her palate 10 years before examination at the clinic. The tumor had recurred 4 years prior to her examination at the clinic, and 2 years before her examination a growth had caused bulging of the ala of the nose. At the time of her examination at the clinic, she had a huge hard growth that involved the entire palate and maxillary sinus and caused bulging of the right eye. Palliative roentgen therapy only was employed.

Of the 3 patients with lymphosarcoma, 2 were males and 1 was a female. Their ages were 1 year, 11 years, and 43 years, respectively. They all had palpable lymph nodes at the time of their examination. All received palliative roentgen therapy with some improvement, but they all died within 11 months, apparently of generalized metastasis.

INTRASPINAL EPIDERMOIDS, DERMOIDS, AND DERMAL SINUSES

CARL FELIX LIST, M.D., Ann Arbor, Michigan

INTEREST in rare diseases is aroused first by detailed case reports, but when the series of observations becomes large and diversified, single cases lose their value as curiosities. One is then tempted to combine the mosaic of individual observations into a general pattern and to outline the salient features of the disease entity in question.

For such an undertaking, I have selected the intraspinal epidermoids, dermoids, and dermal sinuses because of an opportunity to observe personally 5 such cases. These, presented below, will bring the total number reported to 56.

First it will be necessary to define what is meant by the terms "epidermoid, dermoid and dermal sinus," because the terminology on this subject is rather confused. For example, frequent reference is made to the "pearly tumor" which merely describes the shiny macroscopic appearance of some epidermoids, or "cholesteatoma" which represents no anatomical entity, but has been used indiscriminately to designate epidermoids, dermoids, certain mesenchymal tumors and chronic inflammatory conditions.

Epidermoids, dermoids, and dermal sinuses are heterotopic formations composed of elements of the skin. They are dysembryonic malformations (hamartomas) rather than true neoplasms, and are referred to in this paper as tumors because of their expanding nature.

Epidermoids are composed of the superficial (epidermal) layers of the skin. Dermoids contain all layers of the skin, epithelial and mesenchymal, and also the accessory cutaneous organs such as sebaceous glands and sweat glands and hair follicles. Dermal sinuses are tubular formations which may have the histological characteristics of epidermoids, dermoids, or both, but which are always in direct connection with the skin. Thus, a pilonidal cyst is a dermal sinus containing hair. In all three of the above conditions named the viable cells form a thin capsule or wall, and the interior consists of cheesy amorphous masses of dead, cornified epithelial cells, and, in dermoids, also of sebaceous matter and hair. Hair containing tumors have always been classified as dermoids in this study, even though microscopic examination of the cyst wall showed no cutaneous appendages. As Bostroem pointed out, only a small area of the dermoid cyst wall, the "dermoid villus," may possess hair follicles and other accessory cutaneous structures, whereas the remaining portion is purely epidermal in character.

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ILLUSTRATIVE CASES

1. Epidermoids

CASE 1. Low back pain syndrome with right sciatic radiculitis. Myelographic evidence of benign intradural tumor at the level of the third lumbar vertebra. Intra-arachnoid epidermoid removed. Perfect recovery. R. L., No. 459,514, male, aged 14 years, referred by Dr. W. Towsley, Midland, Michigan, admitted to hospital April 10, 1940.

For 7 months prior to admission, the patient complained of increasingly low back pain with radiation along the posterolateral aspect of the right thigh and calf. The pain was sharp, occasionally shooting in character and was increased or elicited by movements, sneezing, laughing, and straining.

On examination, the patient stood with a protective posture, keeping the right leg flexed in knee and hip and slightly externally rotated. The third and fourth lumbar spinous processes were tender to percussion. Considerable spasm of the erector trunci was present. Extension of the lumbar spine was markedly limited and painful, whereas flexion was only moderately limited. The neurological signs were confined to the right lower extremity, except for Lasègue's sign which was strongly positive on the right and suggestive also on the left. There was flabbiness of right gluteus maximus and hypotonicity in the distal segments of the right lower extremity. Slight paresis was present in the gluteus maximus and hamstring muscles on the right. The right knee reflex and hamstring reflexes were diminished and the ankle reflex was absent on that side. Mild tactile and thermic hypesthesia and hypalgesia were demonstrable in the fifth lumbar and first sacral dermatomes. Sphincter functions were unimpaired.

Spinal puncture, done in the lateral recumbent position, revealed colorless spinal fluid under 155 millimeters pressure of water. There was no manometric block. Cell count was 0; Pandy, positive; total protein, 93 milligrams per cent; goldsol, 0012221000; mastic, 2221000; Kahn, negative.

Roentgen-ray examination of the lumbar spine showed no bony changes.

Myelography, first done with air, later on repeated with lipiodol, revealed an identical picture in each (Fig. 2a and b). A smooth ovoid, intradural tumor was outlined posterior to the body of the third lumbar vertebra and intervertebral disc below. The tumor did not obstruct the spinal canal completely.

Diagnosis was made of intradural tumor of the cauda equina at the level of the third lumbar vertebra and intervertebral disc below. A benign lesion such as an epidermoid or dermoid was suspected.

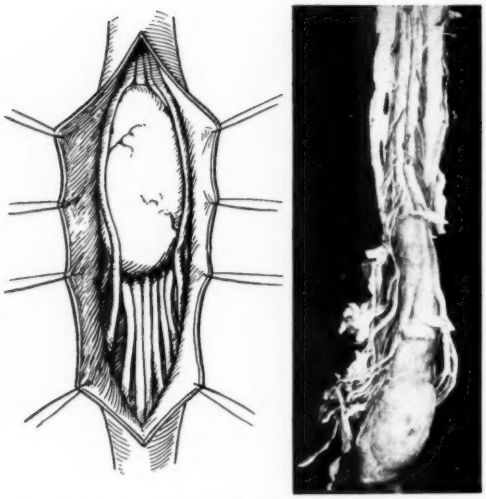

Fig. 1. a, left. Epidermoid of cauda equina as shown at operation. Case 1. b, Dermoid of lumbosacral portion of the cord. Autopsy specimen, reproduced from Pitotti's article.

Laminectomy was performed by Dr. M. M. Peet, on April 19, 1940, under avertin-nitrous oxide anesthesia. The posterior laminae of the second, third, and fourth lumbar vertebrae were removed and the dura was opened. A glistening pearly white tumor, the size of a walnut was found lying between the roots of the cauda equina, covered by arachnoid. The tumor looked exactly like a silkworm cocoon (Fig. 1a). It had a thin, avascular capsule and was removed *in toto* without rupturing it.

Pathological report: epidermoid cyst, filled with keratinized dead epithelial cells.

The postoperative course was entirely uneventful. Check-up examination, 2 months after operation, revealed no abnormal findings except for an absent right ankle reflex. The patient had no complaints.

CASE 2. History of three back injuries. Syndrome of cauda equina lesion. Herniated nucleus pulposus (between third and fourth lumbar vertebrae) or traumatic arachnoiditis was suspected clinically. Myelography revealed tumor between third and fourth lumbar vertebrae. Intra-arachnoid epidermoid found at operation. Removal with practically complete recovery. R. M., No. 452,882, male, aged 47 years, referred by Dr. W. Mertaugh, Sault Ste. Marie, Michigan, was admitted to hospital November 13, 1939.

Five or 6 years prior to admission, the patient suffered a severe back injury in a toboggan accident. For several weeks he had severe backache and pains in both legs. Even after recovery from the acute symptoms, a dull ache in the lumbar spine and pains in the legs persisted, especially on exertion. Four years prior to admission, patient sustained another back injury (automobile accident). He required 5 weeks' hospitalization and a diagnosis of fractured lumbar spine was made. There was in addition radiation of pain along the course of the left sciatic and of the right crural nerve.

Two years prior to admission, the patient suffered a third back injury, when he fell on a steel plate. His left leg became immediately paralyzed for 15 minutes, and he experienced severe pain in the left groin. On admission, the patient complained of persistent backache in the lumbar region, shooting pains radiating to the anterior surface of both thighs, worse on the left, and also of left sciatic pain. The pains were aggravated by movements or by coughing. During the past 8 years increasing constipation had been noticed.

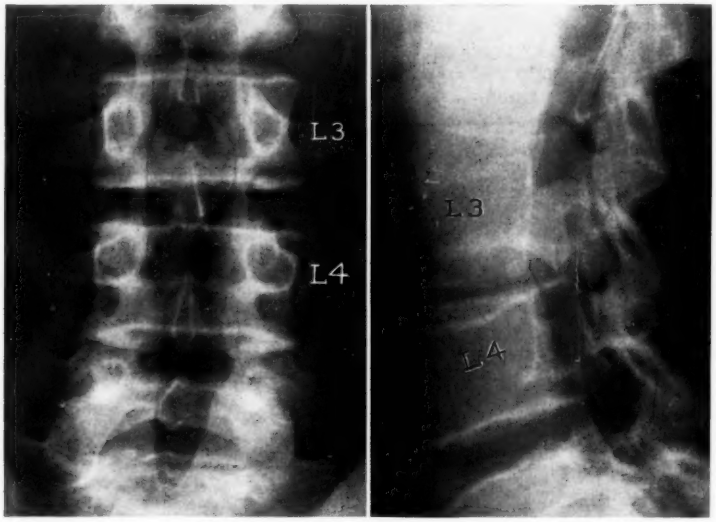

Fig. 2. a and b, Air-myelogram, Case 1. Epidermoid at third lumbar vertebra.

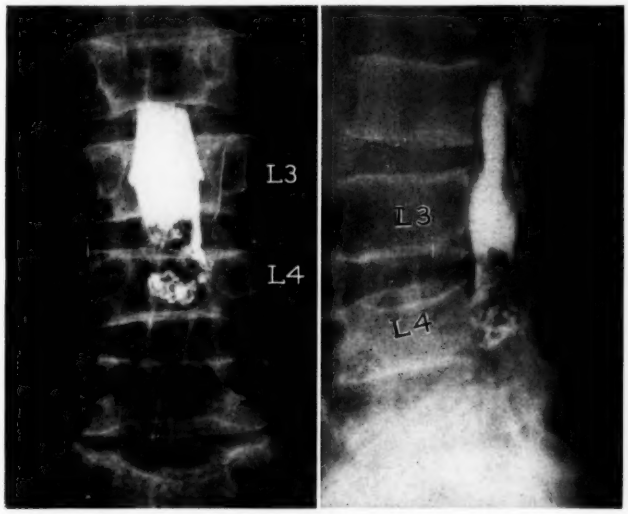

Fig. 3. a and b. Lipiodol-myelogram, Case 2. Epidermoid at third and fourth lumbar vertebrae.

On examination the patient stood with a rigid, anteflexed posture, the normal lumbar lordosis being effaced. There was considerable spasm of the erector trunci bilaterally, with marked limitation of all low back movements. Lasègue's sign was strongly positive bilaterally, and sharp pain was elicited also on stretching the crural nerves. There was paresis of left anterior thigh muscles with considerable diminution of the left knee reflex. All other reflexes were normal. Tactile hypesthesia and hypalgesia were present in the left second lumbar to second sacral dermatomes, and vibratory sensation was diminished in the left foot. The neurological examination was otherwise normal, except for horizontal nystagmus.

Lumbar puncture, done in the lateral position, revealed so low a spinal fluid pressure that it could not be measured nor could the effect of jugular compression be determined. The clear, colorless spinal fluid contained 1 lymphocyte per cubic centimeter; Pandy was strongly positive; goldsol 0001111000; total protein, 108 milligrams per cent.

Roentgen-ray examination of the spine showed hypertrophic arthritic changes in the lumbar region, but no evidence of fracture.

Myelography with lipiodol, injected by lumbar route, showed complete obstruction of the spinal canal at the level of the fourth lumbar vertebra. Above the obstruction the mottled flaky surface of an intradural tumor was outlined which extended upward to the level of the intervertebral disc between the third and fourth lumbar vertebrae. (Fig. 3c and d).

Diagnosis: While clinical history and findings favored the diagnosis of posttraumatic arachnoiditis of the cauda equina or of rather high herniation of the nucleus pulposus on the left (between third and fourth lumbar vertebrae), myelography suggested intradural tumor with associated arachnoiditis. A meningioma was suspected.

Operation was performed by Dr. C. F. List, on November 30, 1939, under avertin-ether anesthesia. The posterior laminae of the third, fourth, and fifth lumbar vertebrae were removed and also the left inferior articular facet of fourth lumbar vertebra, which protruded abnormally into the spinal canal, as if it had been previously fractured and dislocated. The hypertrophied ligamentum flavum between fourth and fifth lumbar and between fifth lumbar and first sacral vertebrae was also excised. When the dura was opened, an intra-arachnoid, ivory white, soft caseous epidermoid, the size of a date was encountered. The tumor extended from the level of the third to fourth lumbar vertebra. Its avascular capsule was exceedingly thin and firmly adherent to roots of the cauda equina on the left.

Fig. 4. Lipiodol myelogram, Case 3. Dermoid extending from twelfth dorsal to second lumbar vertebra. Note enlargement of spinal canal at that level. Arrow points to a markedly atrophic pedicle.

There were many dense arachnoid adhesions even beyond the confines of the tumor. Because the friable tumor capsule could not be separated from the nerve roots without damage, a complete intracapsular removal of the epidermoid contents was performed and then as much of the capsule was resected as possible.

Pathological report: epidermoid consisting of desquamated keratinized epithelium.

Postoperative course was uneventful except for temporary retention of urine, requiring inlying catheter for 2 weeks.

Check-up examination, 7 months after operation revealed the following: The patient stated that his pains disappeared immediately after operation and never returned. He walked, moved about, and even danced without discomfort, but still complained of slight constipation. Examination showed almost normal range of back movements, negative Lasègue, no muscular weakness, but slight atrophy of left thigh. There was marked diminution of the knee reflex and slight diminution of adductor and ankle reflex on the left. Mild hypalgesia was present in the second to fourth lumbar dermatomes on the left.

2. Dermoid

CASE 3. Syndrome of cauda equina lesion with marked signs of local meningeal irritation. Roentgenological evidence of intradural tumor extending from the twelfth dorsal to second lumbar vertebrae. Almost complete removal of partially intramedullary dermoid of cauda equina and conus terminalis. Recovery with residual cauda equina syndrome. Considerable late functional improvement after secondary orthopedic operations. T. A., No. 432,216, female, aged 8 years, referred by Dr. J. T. Jerome, Traverse City, was admitted to the hospital October 13, 1938.

Three years prior to admission, the child complained of occasional pain in her right hip, exaggerated by straining. For more than a year she also experienced dull backaches in the midline of the upper lumbar region. This pain was eased by walking about and aggravated by exertion. Recently it increased in intensity and became almost continuous. In the last months prior to admission, the child assumed a peculiar twisted posture of her back, urination became difficult to start, and there were complaints of constipation and of tingling sensations in both feet.

On examination, the child stood with a rigid lumbar spine and slight list to the right. Severe muscle spasm of the long back muscles prevented flexion and extension movements, especially of the low dorsal and upper lumbar spine, whereas lateral inclination was less limited. There was marked local tenderness of the first and second lumbar spinous processes; coughing, jugular compression or even flexion of the neck produced nonradiating pain in the same location. Some stiffness of the neck was present; this was thought to be a protective muscle spasm to prevent transmission of movement to the upper lumbar spine. The right calf muscles were slightly atrophic. Bilateral mild equinus posture and hypotonicity of the feet were noted. Lasègue's sign was strongly positive on both sides. There was slight weakness of long toe extensors, toe flexors, peronei, and of hamstring muscles bilaterally. Patellar and gluteal reflexes were diminished, ankle, hamstring, and plantar reflexes were absent bilaterally. Sensory or sphincteric disturbances were absent. Bilateral horizontal nystagmus was also noted.

Lumbar puncture, done by the referring physician, revealed a clear colorless fluid under pressure of 205 millimeters water. On jugular compression, there was partial manometric block. Cell count was 31, type of cell not specified; Pandy, strongly positive; and goldsol, 0000012210.

Roentgen-ray examination showed definite increase of the anteroposterior and transverse diameter of the spinal canal, at the level of the twelfth dorsal, first and second lumbar vertebrae. Also the pedicles and posterior margins of the bodies of these vertebrae showed erosion from pressure.

Myelography, with lipiodol, injected by lumbar route, revealed complete obstruction of the spinal canal at the superior margin of the second lumbar vertebra with "cupping" of the lipiodol, suggestive of intradural tumor (Fig. 4).

Diagnosis was made of a slowly growing tumor, located in the conus region and upper portion of the cauda equina, corresponding to the bony level of twelfth dorsal to second lumbar vertebra. The tumor was thought to be a neurinoma or an ependymoma of the filum terminale.

Operation was performed by Dr. S. Braden, on October 22, 1938, under avertin-ether anesthesia. The posterior laminae of the twelfth dorsal, first, second, and third lumbar vertebrae were removed. The first and second lumbar laminae were found to be markedly thinned, the epidural fat absent at this level and the dural sac enlarged. After the dura was opened, a smooth whitish dermoid of sausage shape and the size of a thumb was seen within the arachnoid, displacing the roots of the cauda equina anteriorly. The lower pole of the tumor was free, yet some nerve roots were firmly adherent to its avascular capsule. The dermoid consisted of two main masses which were connected by a narrow bridge of fibrous and adipose tissue. The upper pole of the tumor had an intramedullary location within the conus and epiconus from which structures it could not be dissected without causing damage. Therefore, the capsule of the dermoid was incised and complete enucleation of the sebaceous material containing some hair was performed. The collapsed remnants of the capsule were folded in and sutured to prevent contamination of the subarachnoid space with free sebaceous material.

Histological report: Cyst wall consisting of cornifying stratified squamous epithelium, a few accessory skin structures, dead hairs and desquamated keratin in the lumen—a dermoid cyst.

Postoperative course was stormy with fever and signs of meningeal irritation lasting a week. Complete paralysis of the bladder developed necessitating inlying catheter; the pareses of the lower extremities were increased also. Cystometric examination, one month after operation, revealed a combination of reflex and autonomous bladder.

Check-up examination, 18 months after operation, disclosed a rather marked cauda equina syndrome with greater involvement on the right. There was calcaneus deformity of the right foot, atrophy and weakness of the right toe extensors, gastrocnemius, hamstring muscles, and gluteus maximus. The left knee reflex and both ankle and hamstring reflexes were absent. There was surface hypesthesia and hypalgesia in the fifth lumbar to fifth sacral dermatome on the right. The disturbance of vesical function, characterized as "autonomous neurogenic type" was still present, but the patient kept herself fairly dry. A triple arthrodesis of the right foot with transplantation of peroneal muscles improved the patient's gait.

3. Dermal Sinuses

CASE 4. Thirteen years' history of intermittent weakness of the legs and of an occasionally discharging pilonidal sinus in the upper dorsal region. One brother had a pilonidal sinus of the sacral region. Findings suggested intramedullary spinal cord tumor in the upper dorsal region. Operation revealed dermal sinus above the third dorsal spinous process extending through the dura into the spinal

cord. Complete removal of hair-containing, sebaceous, intramedullary mass, extending from the first to fifth dorsal segment. Marked improvement with mild residual cord symptoms, unchanged after 4 years' observation. D. D. C., No. 381,180, male, aged 19 years, referred by Dr. H. B. Haynes, Lansing, Michigan, was admitted to the hospital May 4, 1936.

At the age of 6, 13 years prior to admission, the patient noted a slight weakness of the right leg. Half a year later, the left leg also became affected and, within a few months, the left leg became weaker than the right one. Twelve years prior to admission, the patient was examined in another hospital. A mild spastic paraparesis of the legs without sensory changes was then found. Roentgenograms of the spine were said to be normal. Spinal fluid examination showed 15 white blood cells (unspecified), increased globulin and mastic reaction was 33100000. Four years prior to admission, the function of the legs improved so remarkably after chiropractic treatment that the patient was not only able to walk well, but also to play football. He merely noticed slight stiffness of the legs on exertion. Two years prior to admission, however, the weakness of the left leg recurred and the right leg became involved in the last few weeks prior to admission. The patient also stated that he could not always tell "where his left foot was," that he had become constipated and had difficulty initiating urination.

Since earliest childhood the patient was known to have a hair-containing dimple in the upper portion of his back. Occasionally, this area discharged and became red and sore to touch, with radiation of pain to the shoulder blades. The soreness disappeared whenever some of the hair tuft was removed. The patient stated that one brother had a pilonidal cyst removed from the sacral region.

Examination revealed a dimple in the midline between the second and third dorsal spinous process, containing a small tuft of hair. Palpation revealed a deep induration which was tender to pressure, extending from the dimple upward and paravertebrally toward the base of the second spinous process which was also tender.

Neurological examination revealed definite paresis of the left intercostal muscles below the third interspace and of the left abdominal muscles which occasionally exhibited peculiar spontaneous clonic contractions. The right upper abdominal reflex was diminished; all others were absent. The left foot assumed a varo-equinus posture. There was moderate spastic paraparesis of the legs. The paresis was of the so called predilection type and more pronounced on the left. The deep reflexes of the lower extremities were exaggerated with clonus, Babinski and Oppenheim bilaterally. Pain and temperature sensations were diminished in the third and fourth dorsal dermatomes bilaterally, and below this level considerable reduction of all modes of sensation was present, especially on the left. Vibratory sensation of the legs and position sense of the toes were diminished on the right and absent on the left. The patient walked with a spastic-atactic gait, the disturbance being greater on the left. There was fall tendency in the Romberg position.

Lumbar puncture, done in the recumbent posture, showed clear xanthochromic spinal fluid with an initial pressure of 165 millimeters water. On jugular compression, there was almost complete manometric block. After removal of 4 cubic centimeters of fluid the pressure dropped to 0. No cell count could be done because of artificial slight admixture of blood. Pandy and Nonne were strongly positive. Goldsol, 022123332100. Mastic, 123321. Kahn, negative.

Roentgen-ray examination revealed definite widening of the spinal canal at the level of the first to third dorsal vertebrae, with thinning of the left pedicles. There was a spina bifida of the first sacral segment.

Myelography, with lipiodol, introduced by lumbar route, showed complete obstruction of the spinal canal at the lower margin of the third dorsal vertebra.

Diagnosis was made of a probable intramedullary cord tumor occupying the upper dorsal segments. The tumor was thought to be a dermoid connected with the pilonidal sinus.

Operation was performed by Dr. M. M. Peet, on May 11, 1936, under avertin anesthesia. The stalk of the pilonidal sinus was found to be surrounded by scar tissue, apparently resulting from previous infection. The sinus tract extended through the intervertebral space between the second and third dorsal vertebrae. After removal of the laminae of the seventh cervical to the fourth dorsal vertebrae the dermal sinus was seen to penetrate dura and leptomeninges and to terminate within the cord (Fig. 5). The widened spinal cord formed a mere shell around a large intramedullary mass consisting of sebaceous material and a few hairs. As far as could be determined, the intramedullary tumor, which measured 10 centimeters in length, was completely removed.

On histological examination, a cross-section of the deeper, extraspinal, portion of the excised dermal sinus (Figs. 6 and 7) showed a central lumen lined with stratified squamous epithelium, hair follicles, and sebaceous glands. The mesenchymal layer of the dermal sinus was surrounded by a covering of meninges, consisting of arachnoid with psammoma bodies and dura. Various parts of the dermal sinus showed infiltration with pyogenic granulation tissue. The intramedullary mass consisted of dead keratinized epithelium and sebaceous material.

Postoperative course was rather stormy for 9 days with temperature elevation to 104 degrees F., but without severe meningeal signs.

Check-up examination. A sweating test (heat method) done 3 months after operation showed marked hypohidrosis below the fourth dorsal segments bilaterally, but relative hyperhidrosis above this level (Fig. 8).

Re-examination, 4 years and 4 months after operation, revealed a residual Brown-Séquard syndrome with mild spastic paresis of the left leg and trunk, disturbance of deep sensation in the left leg and diminution of pain and temperature sensation on the right below the fifth dorsal segment.

CASE 5. Symptoms and signs of acute Bacillus coli meningitis and, later, epidural abscess in the lumbar region. Lumbosacral pilonidal sinus. Operation showed cylindrical body of epidermoid material embedded in the dura, surrounded by epidural and subdural abscess. Open drainage. Recovery with residual cauda equina syndrome. Late improvement after orthopedic operations. D. W., No. 368,169, female, aged 7 years, referred by Dr. G. J. Prout, Saline, Michigan, admitted September 3, 1935.

The child developed severe headaches 3 days prior to admission. The following day there were nausea and vomiting, and retraction of the head also developed.

Examination, on the Contagious Unit of the Pediatric Department, revealed the following: The child was acutely ill with flushed skin. Temperature was 101.4 degrees F.; pulse, 96; respirations, 24. There was stiffness of the neck and back with positive Brudzinski and Kernig signs. Knee and ankle reflexes were diminished.

Lumbar puncture showed the spinal fluid under increased pressure, cell count was 1400, 95 per cent of which were neutrophil leucocytes, 5 per cent lymphocytes. Pandy and Nonne were strongly positive; goldsol, 0012210; mastic, 242110. The smear showed no organisms and the culture no growth.

Diagnosis of acute meningitis was made, the etiology of which was undetermined.

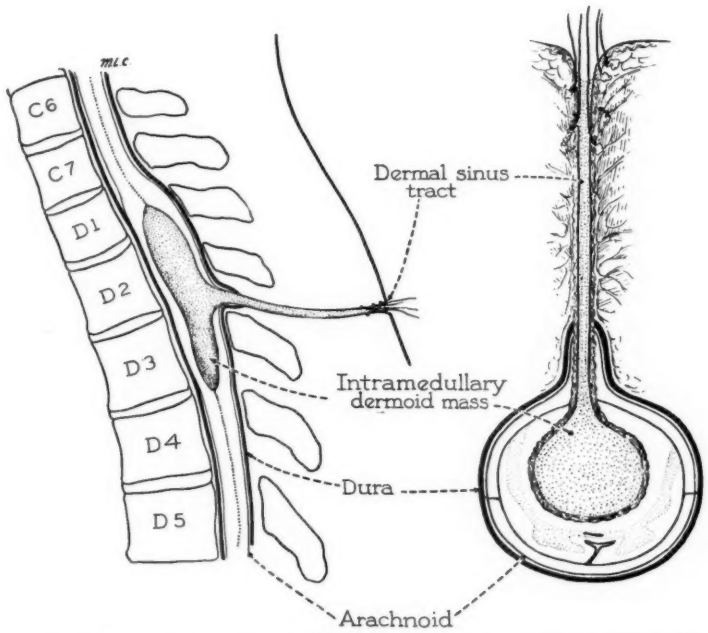

Fig. 5. Case 4. Semidiagrammatic drawing to show the topographic relations of the dermal sinus. At left, midsagittal section; at right, cross-section, showing the meningocele, surrounding the deep portion of the dermal sinus.

Further course in the hospital: The patient's condition grew worse on the second day. The fever rose to 104 degrees F., the pulse to 128, and respirations to 38. The meningeal signs increased. Cisternal puncture then showed 430 polymorphonuclear leucocytes. Culture and smear revealed Bacillus coli and, in the smear only, gram-positive diplococci. Two days later, urinary retention occurred. Again Bacillus coli and diplococci were grown from the spinal fluid, obtained by lumbar tap. In the following 2 weeks, an irregular fever of 101–102 degrees F. and signs of meningitis persisted. Repeated cell counts of the spinal fluid showed a slight drop of cells (average 280) with a greater percentage of lymphocytes. Colon bacilli were repeatedly demonstrated, not only in the spinal fluid, but also in the catheterized urine.

On the twentieth day of hospitalization, the temperature again rose to 103.4 degrees F. When lumbar puncture was attempted between the second and third lumbar vertebrae, 3 cubic centimeters of foul smelling pus was evacuated before the dura was penetrated, culture of which again showed Bacillus coli. Cisternal puncture on the same day yielded clear fluid, containing only 65 white cells, 50 per cent of them being neutrophils and 50 per cent lymphocytes.

Diagnosis of epidural abscess in the lumbar region was made and the patient was transferred to the neurosurgical service. On questioning, the child had complained of numbness of the legs and abdominal distention for the past 2 days.

Repeated neurologic examination showed, in addition to the marked meningeal signs, paresis of the distal segments of both lower extremities with loss of ankle reflexes but no sensory changes could be demonstrated. Another,

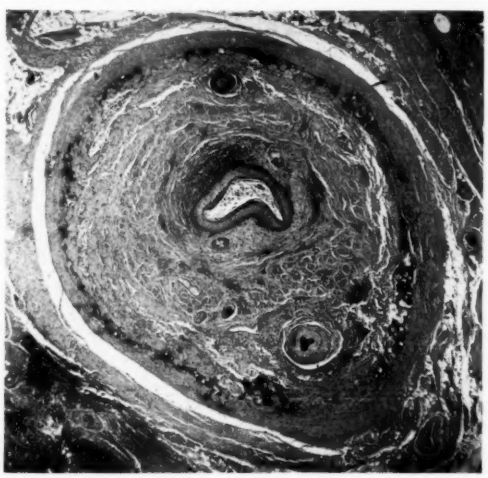

Fig. 6. Case 4. Photomicrograph of cross-section, deep portion of dermal sinus. General view at low power.

finding was considered of significance: In the midlumbosacral region, a small reddened dimple was present, with a tuft of hair, apparently an infected pilonidal sinus.

It was then suspected that the dermal sinus tract extended into the vertebral canal, thus serving as portal of entrance for the infection of the epidural and subarachnoid space.

Operation was performed by Dr. S. Braden on the same day—September 3, 1935—under nitrous oxide anesthesia. As the lumbar muscles were stripped from the spine, foul-smelling pus was encountered at the level of the third lumbar vertebra. Laminectomy of the second to fourth lumbar vertebrae disclosed a large epidural abscess. Two small cylindrical bodies were then seen embedded in the dura; they had a shiny surface with a pearly luster and consisted of soft cheesy material (Fig. 9). The operative note mentions no connection of these structures with the cutaneous dimple. After removal of the epidermoid bodies, pus escaped also from within the dura. After the dural opening was enlarged, a subdural abscess was exposed, the cavity of which measured 5 centimeters in length. The thickened and inflamed arachnoid was left intact. The wound was left open and packed with gauze soaked with metaphen in oil.

On microscopic examination, the cylindrical pearly structures consisted of dead lamellated cornified epithelium showing at one area a narrow lining of viable epithelial cells and polymorphonuclear leucocytes—an infected dermal sinus.

Immediately after operation the temperature returned to normal. Within 2 months the operative wound healed by granulation. The child developed a "cord bladder" with incontinence, saddle anesthesia, and a left talipes equinovarus, which was corrected by subsequent orthopedic operations.

Check-up examination, 4 years and 7 months after laminectomy, showed the child to be in good general health. Function of the left foot was much improved. There was still a "neurogenic bladder" with urinary infection, but the patient could keep herself fairly dry.

Fig. 8. Case 4. Sweating test (iodine-starch method) shows hypohidrosis below the third dorsal segment.

Fig. 7. Higher magnification. *e*, Epidermal layer; *c*, lermal layer, with cellular infiltration; *g*, sebaceous gland; , arachnoid; *pb*, psammoma bodies; *d*, dura.

ANATOMICAL AND EMBRYOLOGICAL CONSIDERATIONS

Survey of the literature and of the aforementioned 5 cases reveals a total number of 56 cases, viz. 23 cases of single epidermoid, 19 cases of single dermoid, 3 cases of multiple epidermoids and dermoids, and 11 cases of dermal sinus[1] with intraspinal extension.

This group, taken as a whole, shows preferential location in the lower portion of the spinal

[1]The most common variety of dermal sinus (pilonidal sinus or cyst), which is situated in the sacrococcygeal region and usually has no meningeal connection, is considered beyond the scope of this paper.

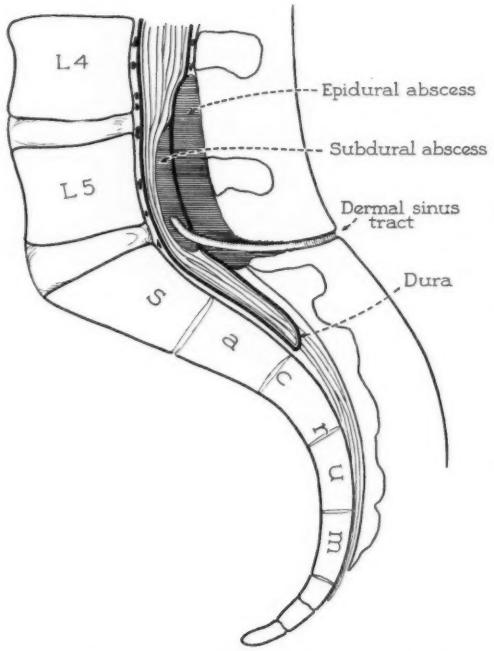

Fig. 9. Semidiagrammatic drawing of dermal sinus, Case 5, to show epidural and subdural abscess. The epidural abscess was more extensive than pictured here.

canal: 35 cases, below the twelfth dorsal vertebra, 62 per cent. Only in one single instance (Mixter), the lesion originated in the cervical spine; in another exceptional case (Ivanoff), it extended throughout the entire length of the cord up to the medulla oblongata.

More pertinent information is obtained by considering the three types of dermal formations individually, and by dividing them into lesions of (1) the cervical cord, (2) the upper thoracic portion of the cord, (3) the lower thoracic cord, (4) the lumbosacral cord—epiconus and conus—and (5) the cauda equina and filum terminale (Fig. 10). Epidermoids are then found to exhibit fairly uniform distribution over the upper dorsal, 4 cases; low dorsal, 6 cases; and lumbosacral portion of the cord, 5 cases; but their incidence is somewhat higher at the level of the cauda equina, 8 cases. Dermoids show a striking preference for the lumbosacral portion of the cord, 12 cases, (see Fig. 1b), but are less commonly confined to the cauda equina, 4 cases. They are rare in other locations: cervical, 1 case; upper thoracic, 2; low thoracic, 1. Dermal sinuses are found either in the upper thoracic area, 4 cases, or in the distal parts of the cauda equina, lumbosacral portion of the spine, 6 cases, whereas they appear to be rare at other levels, 1 case in low thoracic region.

An attempt was made to group these lesions according to their relationship to the meninges as extradural, subdural, subarachnoid, subpial and intramedullary. Unfortunately, such classification must remain inaccurate, because descriptions in the literature sometimes lack detail, or because it may be impossible to determine the exact origin of a large tumor. Giving due consideration to these reservations, it may be stated that almost two-thirds of all lesions were situated within the leptomeninges, one-fifth frankly intramedullary and the few remaining ones confined to the extradural space. Epidermoids, especially those occurring in the thoracic region, were often found to be intramedullary. Dermal sinuses infrequently penetrate as far as into the cord itself, but usually end in the epidural or subdural spaces. It may be mentioned here, that the superficial parts of a dermal sinus assume the histological characteristics of external skin, whereas the deep cul-de-sac may possess no cutaneous appendages and hence resemble an epidermoid.

Most lesions exhibited preference for the posterior circumference of the cord, a fact of pathogenetic significance: cases of Fraser, Schroeder, Harriehausen, Michelsen, Mixter, and others.

There exist three observations of multiple epidermoids and dermoids within the spinal canal: Trachtenberg, Raymond, Alquier, and Courtellemont, and Muscatello. Bostroem and others described an occasional tendency of both dermoids and epidermoids to form "daughter" nodules, presumably due to rupture of the thin capsule of the main mass. Such rupture of the growth probably caused widespread dissemination of cholesteatomatous material over the entire subarachnoid space in Trachtenberg's case—primary dermoid of the lumbosacral cord—and Raymond, Alquier, and Courtellemont's case—probably primary dermoid of the frontal lobe. On the other hand, epidermoids and dermoids may be primarily multiple as in Muscatello's case which will be discussed later.

Embryological investigation furnishes the clue to the pathogenesis of epidermoids, dermoids, and dermal sinuses. A generally accepted theory (Bostroem, Critchley and Ferguson, Walker and Bucy) assumes that these formations arise from displaced cutaneous-ectodermal cell inclusions. This heterotopia is thought to occur in early fetal life, probably in the third to fifth week. During this period, the neural groove closes to form the

medullary tube and the ectoblast differentiates into cutaneous and neural ectoderm. Cutaneous ectodermal cells may then be trapped either within the neural tube or near the closing medullary folds and thus become the first *anlage* of intramedullary or paramedullary epidermoids or dermoids. If the cleavage of cutaneous and neural ectoderm remains incomplete at a certain point, a dermal sinus will develop connecting the external skin with the neuraxis. This theory is well supported by von Verebely's case. There was a large extradural tumor in the lumbar portion of the spinal canal, consisting of 2 separate cysts. The inner cyst was neuroepithelial in type, betraying its origin from the neural ectoderm; the external cyst, however, was a dermoid derived from the cutaneous ectoderm.

Whether a dermoid or epidermoid will develop depends, in the opinion of most authors, upon the period during which the cell inclusion occurred. If it happened very early in fetal life, the displaced cells are still completely potential and able to form a dermoid; if, at a later period, the cells have already undergone partial differentiation and become unipotential, an epidermoid will develop. Other authors believe that a dermoid results from displacement of cells belonging to all cutaneous layers, whereas an epidermoid is derived from epidermal cells only. Strictly speaking, dermoids may be considered as bigerminal teratoid formations. There exist also transitional forms between dermoids and true teratomas.

Knowing their mode of origin, we now understand why epidermoids, dermoids, and dermal sinuses so frequently occur in the midline or at the posterior circumference of the cord. Evidently, these lesions are associated with a disturbance in the "posterior closure line" of the neuraxis and sometimes of the vertebral canal as well. A few examples, selected from the literature, will show that the dermal formations under discussion may be classified with the so-called dysraphic disorders. In Harriehausen's and Hamby's cases a posteriorly situated dermoid of the lumbosacral cord marked the beginning of a complete diastematomyelia. In Fraser's and in Salotti's cases, tumors in the posterior median sulcus produced a deep groove which, according to these authors, suggested rudimentary duplication of the cord. Most instructive of all is Muscatello's case. His patient showed a wide posterior rachischisis of the third to tenth thoracic and fourth and fifth lumbar vertebrae. An extradural dermoid was present at the level of the fourth thoracic vertebra, and a small epidermoid at the level of the fourth and fifth lumbar vertebrae. Furthermore, there was hydromyelia and diastematomyelia. This case, then, demonstrates a combination of multiple dysraphic disorders of the cord and spine with two congenital tumors. It is also worthy of mention that spina bifida occulta is frequently present at the level of a dermal sinus: cases of Walker and Bucy, Ottonello, Ripley and Thompson, Moise, Sharpe, and Hipsley. The dysraphic character of the dermal sinus is clearly shown in our Case 4, even though no spina bifida existed. Here, the deeper portion of the sinus tract was surrounded by a meningeal covering (meningocele) containing dura and leptomeninges with psammoma bodies (Figs. 6 and 7).

Recent embryological work dealing with the development of the lower part of the spinal cord (Holmdahl) and of the filum terminale (Streeter) may give some explanation as to why dermoids and also epidermoids occur preferentially in the distal portion of the neuraxis. Holmdahl casts aside the orthodox view that the neural tube is exclusively formed by ectoderm. He points out that the caudal part of the neural tube develops directly from an undifferentiated cell mass not belonging to any particular germinal layer. According to this author, the low thoracic and entire lumbosacral portion of the human cord are formed in this manner, which he terms "secondary phase of body development" in contrast to the primary phase of development from germinal layers. Holmdahl's findings would readily explain the relative frequency of dermoids and other dysembryonic tumors (teratomas) in the lumbosacral portion of the cord, since one has only to assume that disorderly differentiation of the primitive matrix causes these tumors.

Streeter's work on the development of the filum terminale is based on the fact that the vertebral column grows much more rapidly than the spinal cord. As a result, the distal end of the cord finally corresponds to the upper border of the second lumbar vertebra. The coccygeal segments of the cord become elongated to the filum terminale and the nerve roots to the cauda equina. A process of dedifferentiation transforms the coccygeal segments into the filum terminale. Since the distal—coccygeal—end of the cord is originally attached to the cutaneous ectoderm, it is evident that dermal inclusions may easily occur at that point. In later phases of embryonic development, the heterotopic dermal material keeps its close topographic relation to the distal end of the cord—filum terminale—and hence becomes situated in the upper lumbar portion of the vertebral canal, a preferential location for both dermoids and epidermoids. The presence of dermal inclusions dis-

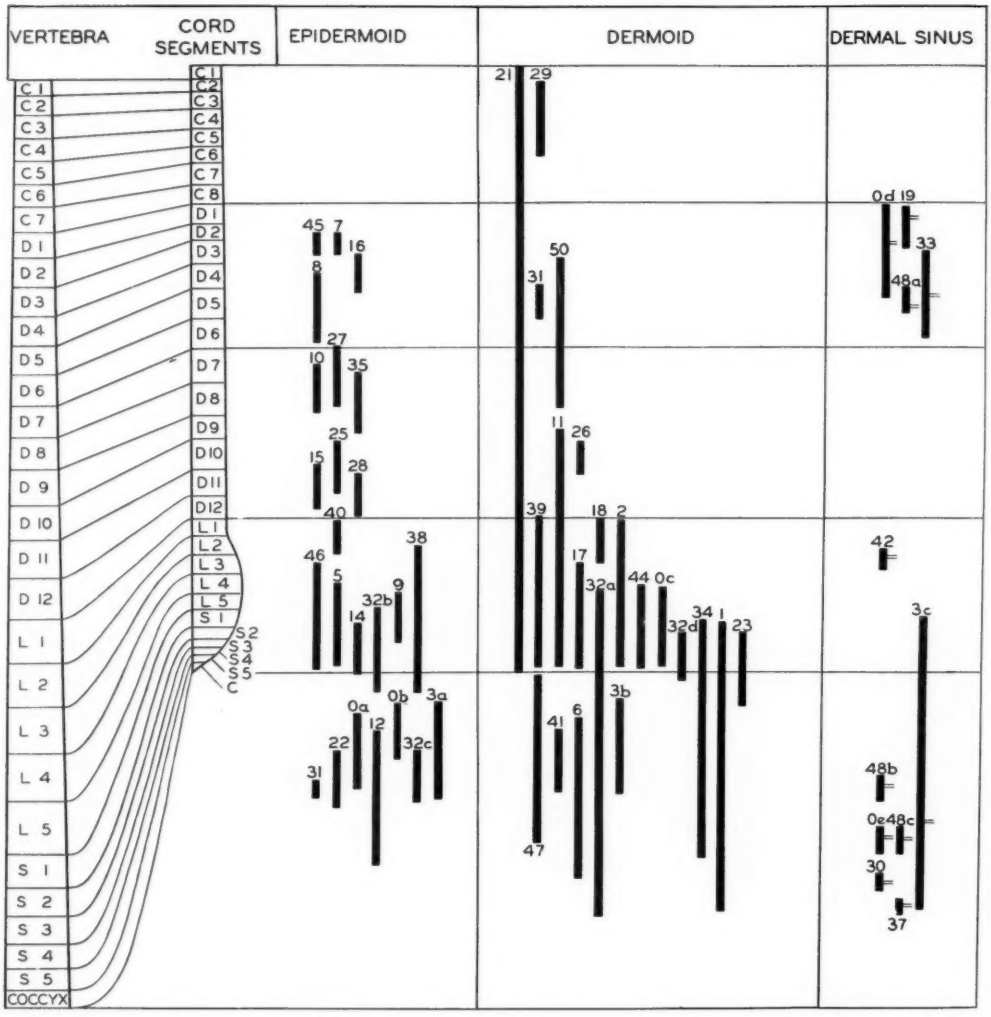

Fig. 10. Diagram to show the location and size of epidermoids, dermoids, and dermal sinuses, in relation to the spinal cord segments and to the vertebrae. Numbers indicate the authors which appear in the appended bibliography; letters after the numbers are used when the author described several cases. *O*, *a* to *e* designates our own cases. Cross-bars in the group of dermal sinuses indicate the level of the sinus tract.

turbs the normal dedifferentiation of the coccygeal segments to the filum terminale: thus, in 2 cases of dermoid (Bradford, Pitotti) (Fig. 1b), the sacral portion of the cord, involved by tumor, actually extended down to the sacrum as it does in early fetal life. In Boldrey and Elvidge's second case, there was anomalous dilatation of the ventriculus terminalis above the dermoid.

CLINICAL AND DIAGNOSTIC CONSIDERATIONS

Incidence as to sex and age, duration of symptoms. Epidermoids appear to be more frequent in the male—15 male, 8 female, whereas dermoids and dermal sinuses are about equally distributed in both sexes—16 male, 14 female.

It is customary to chart a disease according to its incidence in various age groups. That method

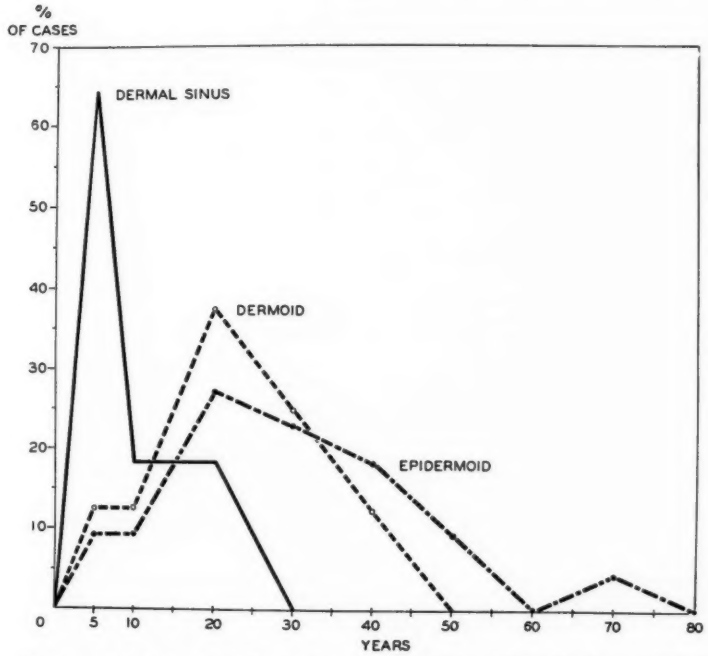

Fig. 11. Graph to show the frequency with which epidermoids, dermoids, and dermal sinuses become clinically manifest in various age groups.

is apt to be misleading in epidermoids and dermoids, because these extremely slow growing tumors are present, though latent, from birth. They may come under observation at any stage, even before causing clinical symptoms; they were accidental autopsy findings in the cases of Muscatello and Lauterburg. Their natural course may last over the entire span of life. It is, therefore, preferable to group them according to the age at which the first clinical symptom appeared. As shown in the graph (Fig. 11), the majority of dermal sinuses become clinically manifest within the first decade, in fact usually before the age of 5, because of their tendency to become infected. Dermoids cause their first symptoms within the first two decades, especially in the late adolescent period. Epidermoids are slightly slower to develop; as a rule they become manifest in the early adult period, but may cause their first symptoms any time between the ages of 20 and 50. After the age of 50, both epidermoids and dermoids rarely occur, yet in a patient of Bouchut, Dechaume and Michailidis, the first symptom was noted at 61; death at 71. The average age of 22 patients with epidermoids was 29.8 years, their first symptom occurred at 23.5 years, hence the observed duration of symptoms was 6.3 years. In 16 dermoids the average age was 28.3 years, the first symptom occurred at 18.7 years, and the duration was 9.6 years. These figures emphasize the extremely slow rate of growth in both groups. A course over 10 years is not unusual, and in 2 instances—Case 1 of Naffziger and Jones, and in the case of Verbiest the patients were known to have shown symptoms for 42 and 43 years, respectively. That dermoids become manifest sooner than epidermoids might be due to the fact that dermoids expand by secretion of sebaceous glands in addition to the mere accumulation of dead epithelial cells which is typical for epidermoids.

A peculiar feature of spinal epidermoids and dermoids is their tendency to long remissions. There were 12 cases with long asymptomatic periods. In Verbiest's case, for example, remission lasted 22 years; in other instances, 10 and 8 years. It is difficult to explain the free interval, as sudden gross variations in size of the tumor are unlikely. Marked improvement during the period of remission may mislead the clinician and cast doubt on the diagnosis of cord tumor.

Neurological symptoms. As to neurological symptoms and signs, epidermoids and dermoids differ little from other slowly growing tumors within the spinal canal. The clinical symptomatology largely depends upon the level and extent of the lesion. As in other slowly growing neoplasms, the degree of objective motor and sensory impairment may be amazingly slight in comparison to the magnitude of anatomical damage.

Lesions implicating roots of the cauda equina are often characterized by exceedingly severe signs of radicular and local meningeal irritation which may entirely dominate the clinical picture (Naffziger and Jones, Bagley and Arnold, and my own observation). These patients exhibit marked spasm of the long back muscles with bizarre antalgic postures. Kernig's, Lasègue's, and Brudzinski's signs are strikingly positive. Typical root pain can be elicited by slight jugular compression or movements of the head, especially flexion (Naffziger and Jones, and my own observation). The unusual severity of root pain observed in epidermoids and dermoids has been attributed to chemical arachnoiditis. Considerable local irritation and inflammatory changes are presumably caused by cholesterol crystals (Mahoney) or free fatty acids (Verbiest) contained in these tumors.

In lesions of the thoracic cord, radiculomeningeal symptoms are less prominent; a transverse syndrome suggestive of intramedullary process is often present, since a high percentage of thoracic epidermoids actually are intramedullary. Occasionally, striking disturbances of vegetative functions such as disorders of sweating (our case 4, Fig. 8) may be associated with intramedullary lesions of the thoracic cord.

Dermal sinuses deserve special comment because of their highly characteristic symptomatology. A telltale dimple in the posterior midline, containing a tuft of hair and occasionally discharging sebaceous material, establishes the diagnosis, especially, when neurological symptoms point to the same level. The skin lesion, however, may be so inconspicuous that it is overlooked until the dermal sinus has become infected. Then, there is pain and local inflammatory infiltration and the dimple discharges purulent material. When infection reaches the deeper portion of the sinus, symptoms and signs of epidural or subdural abscess and of meningitis may develop (Walker and Bucy and others). In etiologically obscure cases of meningitis or spinal epidural abscess, therefore, one should always consider the diagnostic possibility of infected dermal sinus as portal of entry for the infection (Moise, Ripley and Thompson, and others). Dermal sinuses of the lumbosacral area frequently become infected in early childhood, since that region is apt to be contaminated with Bacillus coli, pyogenic cocci, and other organisms. Sometimes the patients pass through recurrent episodes of meningitis (Walker and Bucy). Dermal sinuses of the upper dorsal area are less often infected, but since their narrow external opening may easily be obliterated, sebaceous matter continues to accumulate in the deep intraspinal portion of the sinus with resultant compression of the cord.

Spinal fluid findings. The changes of spinal fluid found in epidermoids and dermoids do not differ essentially from those of other cord tumors. Pleocytosis is absent even in cases with marked meningoradicular signs. Although the amount of total protein may be considerably increased, it rarely reaches the excessive levels commonly found with neurinomas. In 3 cases in the literature, cholesteatomatous material was obtained by lumbar puncture, thereby verifying the lesion. In Raymond, Alquier, and Courtellemont's case, presumable rupture of a dermoid accounted for generalized dissemination within the subarachnoid space. In a second case, reported by Forrestier, Haguenau, and Petit-Dutaillis, an epidermoid at the level of the first and second lumbar vertebrae was accidentally punctured on attempted spinal tap. The same thing happened in a case of lumbar dermoid reported by Boldrey and Elvidge.

Cases of infected dermal sinus show various types of meningitic changes in the spinal fluid. The fluid may be actually infected or sterile, and pleocytosis may be polymorphonuclear or lymphocytic, according to the stage of infection.

Roentgenographic findings. Roentgenograms of the spine may reveal widening of the spinal canal at the level of the lesion with atrophy of the pedicles and laminae from pressure. This change possibly is more frequent in the expansile dermoids than in the softer and more easily molded epidermoids; on the other hand, the degree of bony change may depend mainly upon the duration of the lesion.

Myelography is not only helpful in determining the level and size of the tumor, but may sometimes demonstrate finer anatomical details. Thus in Case 1 (Figs. 2a and b), the perfectly smooth ovoid appearance of the tumor suggested the diagnosis of dermoid or epidermoid, because the only moderate increase of total protein seemed to rule out the presence of a neurinoma. In Case 2 (Figs. 3a and b), the mottled and flaky surface of the tumor was visualized by lipiodol. The picture resembled the encephalographic appearance of

cerebral epidermoids, described by Dyke and Davidoff. The contrast medium within the subarachnoid space had apparently filled all the small crevasses and clefts of the lamellated surface of the growth.

TREATMENT

As epidermoids and dermoids are benign encapsulated tumors, permanent cure should be expected following their surgical removal, yet results obtained in cases published thus far have been encouraging only in the past 15 years. In most patients mentioned in the older literature, no surgery was done or it was attempted too late. Results have improved remarkably, when patients were operated upon at an early stage and modern neurosurgical methods used, but even now certain factors may prevent complete success. The desirable total removal of the tumor, which alone insures permanent cure, is not always practicable because the thin capsule, which contains viable cells, may be too friable and firmly adherent to the cord and nerve roots. It may be wiser, therefore, in some cases to leave fragments of the capsule behind in order to avoid injury to nervous structures, even though this may mean future recurrence. Another technical difficulty lies in the total removal of intramedullary epidermoids and dermoids. When dealing with such a condition, surgeons are inclined to be conservative for fear of damaging the cord and hence the extirpation may have to remain incomplete. Nevertheless, even an intrascapular enucleation plus partial removal of the capsule may accomplish long lasting symptomatic improvement. Care should be taken not to contaminate the subarachnoid space with sebaceous material as this may cause considerable meningeal irritation.

When dealing with dermal sinuses, the surgeon is confronted with different problems. The sinus tract should be totally excised. Infected dermal sinuses with complicating epidural or subdural abscess or meningitis require open surgical drainage in addition to removal of the sinus. This procedure is usually followed by rapid disappearance of serious symptoms, even though there may be temporary drainage of spinal fluid through the infected field. Fortunately, the accompanying meningitis is often local and sterile in character and subsides soon after drainage. The surgical treatment of infected dermal sinus is effectively assisted by use of sulfanilamide, sulfathiazole, and similar drugs.

SUMMARY

After a discussion of the terminology, 5 cases of intraspinal dermal formations were reported, viz., 2 epidermoids, 1 dermoid, and 2 dermal sinuses, bringing the total of similar observations to 56.[1]

Epidermoids show a fairly uniform distribution over the thoracic and lumbosacral portion of the cord, but are more frequent in the cauda equina. Dermoids have a definite preference for the lumbosacral portion of the cord. Dermal sinuses chiefly occur in the upper thoracic region or in the lowermost portion of the cauda equina. It is exceedingly rare for any of these lesions to be situated in the cervical region.

Approximately one-fifth of all dermal formations are intramedullary, two-thirds are located within the leptomeninges and the few remaining cases are extradural. Epidermoids of the thoracic cord are frequently intramedullary.

There are rare cases of multiple epidermoids and dermoids, either due to primarily multiple origin or due to implantation metastases in the subarachnoid space from rupture of the main nodule.

The pathogenesis of intraspinal epidermoids, dermoids, and dermal sinuses was discussed with reference to various embryological theories. The relationship of these lesions to the "dysraphic state" was emphasized.

Intraspinal epidermoids appear to have a higher incidence in the male, whereas dermoids and dermal sinuses show approximately equal distribution among the sexes.

On the average, dermal sinuses become manifest in childhood, dermoids in adolescence—before the age of 20—and epidermoids cause their first symptoms in early adult life, after the age of 20.

The clinical course of all these lesions is long, sometimes lasting over several decades, and may be interrupted by long remissions.

The neurological symptomatology depends mainly upon location and size of the lesion. If nerve roots of the cauda equina—are implicated, signs of local meningeal and radicular irritation may be very severe, presumably due to accom-

[1]After completion of this paper, the following 4 additional cases reported in the literature came to my attention and I also had an opportunity to observe another case myself, thus bringing the entire number of observations to 61.

King reported a rather typical case of epidermoid of the cauda equina. Stammers described 2 cases of infected dermal sinus, 1 in the lumbosacral the other in the sacrococcygeal area; both showed evidence of meningitis. Hamby published an unusual case of dermal sinus at the level of the third lumbar vertebra, associated with duplication of the lower end of the spinal cord. My personal observation concerned a woman, aged 44 years, who was known to have weakness and deformity of the feet for at least 30 years. After her symptoms had been stationary for 25 years she finally developed a full blown conus syndrome. Lumbar puncture revealed a manometric block on jugular compression and xanthochromic fluid with moderate increase in total protein. The spinal canal was widened at the level of the first and second lumbar vertebrae. At operation, an intramedullary dermoid of the lumbosacral cord was found which had formed multiple intramedullary "daughter" nodules. All these additional cases confirm the statements made in the body of this paper and bring out no further points of special interest. References of the above authors are appended to the bibliography.

panying chemical arachnoiditis. Dermal sinuses are easily recognized by their typical clinical picture. They tend to become infected with resultant epidural and subdural abscess and meningitis.

Changes of the spinal fluid, found in the various dermal formations, are similar to those observed in other intraspinal tumors, although the increase of total protein appears to be less marked than with neurinomas. In rare instances the lesion was directly verified by aspiration of cholesteatomatous material at lumbar puncture. In cases of infected dermal sinus, the spinal fluid may show various types of meningitic change.

Roentgenographic changes of the spine are observed, consisting of widening of the spinal canal. This apparently is more common in dermoids. Spina bifida is frequently found at the level of a dermal sinus. Myelography not only determines the location and size of these lesions, but may give some hint as to the anatomical nature.

The results of surgical treatment of intraspinal epidermoids, dermoids, and dermal sinuses are good, provided they are completely removed. If accompanying arachnoiditis or intramedullary location make such procedure impracticable, intracapsular enucleation may assure long lasting relief. Infected dermal sinuses require excision and open surgical drainage.

REFERENCES

1. Bagley, C., Jr., and Arnold, J. S., Jr. Am. Neurol. Ass., June, 1940.
2. Berka, F. Centralbl. f. allg. Path. u. path. Anat., 1906, 17: 490–491. Časop. lek. česk., v. Praze, 1906, 45: 332–336.
3. Boldrey, E. B., and Elvidge, A. R. Ann. Surg., 1939, 110: 273–284.
4. Bostroem, E. Centralbl. f. allg. Path. u. path. Anat., 1897, 8: 1–98.
5. Bouchut, J., Dechaume, and Michailidis. Lyon méd., 1932, 150: 694–697.
6. Bradford, F. K. Ann. Surg., 1938, 107: 107–115.
7. Cames, O., Cid, J. M., and Parachu, L. Bol. Soc. de cirug. de Rosario, 1935, 2: 154–162.
8. Chiari. Prag. med. Wchnschr., 1883, 8: 378–380.
9. Craig, J., and Mitchell, A. Arch. Dis. Childhood, 1931, 6: 11–16.
10. Critchley, M., and Ferguson, F. R. Brain, 1928, 51: 334–384.
11. Dal Bò. Dermoid of spinal cord. Seventh Congress of Neuropathology, Torino, 1926. Quoted by Salotti.
12. Delrez, L. Liège Med., 1929, 22: 1667–1675.
13. Dyke, C., and Davidoff, L. M. Bull. Neurol. Inst., New York, 1937, 6: 489–493.
14. Forrestier, J., Haguenau, J., and Petit-Dutaillis. Guérison, Rev. neurol., 1931, 38: 469–473.
15. Fraser, J. Surg., Gynec. & Obst., 1930, 51: 162–168.
16. Gross, S. W. J. Nerv. & Ment. Dis., 1934, 80: 274–284.
17. Guizetti, P. Sistema Nervoso, Trattato di Anat. Patologica per medici e studenti. Edited by P. Foa. Part 10, 1, 187. Torino, 1924.
18. Harriehausen. Deutsche Ztschr. f. Nervenh., 1909, 36: 268–284.
19. Hipsley, P. L. Australian & New Zealand J. Surg., 1933, 2: 421.
20. Holmdahl, D. E. Arch. f. Entwcklngsmechn. d. Organ, 1933, 129: 206–254.
21. Ivanoff, N. S. Quoted by Lauterburg.
22. Lauterburg, W. Arch. f. path. Anat., 1923, 240: 328–352.
23. Love, J. G., and Kernohan, J. W. J. Am. M. Ass., 1936, 107: 1876–1883.
24. Mahoney, W. Ztschr. f. d. ges. Neurol. u. Psychiat., 1936, 155: 416–471.
25. Marinesco, G., and Draganesco. Rev. neurol., 1924, 31: 338–355.
26. Masson, C. B. Arch. Neurol. & Psych., 1938, 40: 554–562.
27. Melnikoff-Raswedenkoff, N. Arch. f. path. Anat., 1931, 279: 702–723.
28. Michelsen, J. Deutsche Ztschr. f. Nervenh., 1932, 127: 123–130.
29. Mixter, W. J. Spinal column and spinal cord, Chap. 3, pp. 69–71, in Vol. 12 of Surgery by Dean Lewis. Hagerstown, Md.: W. F. Prior Co., 1932.
30. Moise, T. Surg., Gynec. & Obst., 1926, 42: 394–396.
31. Muscatello, G. Arch. f. klin. Chir., 1894, 47: 257–301.
32. Naffziger, H. C., and Jones, O. W. Arch. Neurol. & Psychiat., 1935, 33: 941–958.
33. Ottonello, P. Riv. di. patol. nerv. & ment., 1933, 41: 512–531.
34. Pitotti, P. Riv. di. patol. nerv. & ment., 1930, 35: 36–65.
35. Puech, P., Plichet, A., Visalli, F., and Brun, M. Guérison, Rev. neurol., 1936, 66: 56–62.
36. Raymond, F., Alquier, L., and Courtellemont, V. Rev. neurol., 1904, 12: 635–636.
37. Ripley, W., and Thompson, D. C. Am. J. Dis. Child., 1928, 36: 785–788.
38. Robertson, W. E., and Ingham, S. D. Pennsylvania M. J., 1916, 19: 408–413.
39. Salotti, A. Arch. ital. di chir., 1927, 19: 135–157.
40. Schroeder, A. H. An. Fac. de med. de Univ. de Montevideo, 1932, 17: 591–621.
41. Shallow, T. A. Surg. Clin. N. America, 1928, 8: 885–899.
42. Sharpe, W., and Sharpe, N. Neurosurgery. Pp. 369–371. Philadelphia and London: J. B. Lippincott, 1928.
43. Streeter, G. L. Am. J. Anat., 1919, 25: 1–11.
44. Trachtenberg, M. A. Arch. f. path. Anat., 1898, 154: 274–291.
45. Veraguth, O. Ztschr. f. d. ges. Neurol. u. Psychiat., 1934, 150: 579–587.
46. Verbiest, H. Zentralbl. f. Neurochir., 1939, 4: 129–141.
47. von Verebely, T. Arch. f. path. Anat., 1913, 213: 541–545.
48. Walker, A. E., and Bucy, P. Brain, 1934, 57: 401–421.
49. Weeder, S. D. Ann. Surg., 1933, 98: 385–393.
50. White, W. H., and Fripp, A. D. Tr. Clinic. Soc. Lond., 1900, 33: 140–145.

Additional References

1. Hamby, W. B. Arch. Path., 1936, 21: 831–838.
2. King, J. E. J. Ann. Surg., 1939, 109: 649–688.
3. Stammers, F. A. R. Brit. J. Surg., 1938, 28: 366–374.

APPENDICULAR OBSTRUCTION: ITS CLINICAL AND PATHOLOGICAL ASPECTS

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MANY writers, including Van Zwalenburg (6), Wilkie, and more recently Wangensteen and Bowers, have stressed the importance of acute appendicular obstruction as a cause of appendicitis. To be considered a distinct and significant entity appendicular obstruction should possess a characteristic pathological picture and should manifest suggestive clinical symptoms important to the proper diagnosis and prognosis in an individual case. A detailed study of such cases is therefore pertinent to a true evaluation and correlation of clinical evidence and actual pathological findings. This study is based on a group of 70 cases of clinically acute appendicitis in which a factor of obstruction was definitely present. Subjective complaints were determined in most instances by personally questioning the patient several days after operation and the results were compared with the symptoms noted in the preoperative records so as to make the story thorough and complete. The surgeon's description of the appearance of the operative field was considered the most important estimate of the gross pathological state of the appendix. The majority of these appendectomies were performed by one surgeon who was especially interested in the problem. Gross and microscopic examinations of the appendices were made proximal to, at the site of, and distal to the site of obstruction.

The evidence to be presented in this clinicopathological study will support the contention that acute appendicular obstruction is a distinct pathological entity differing from acute appendicitis that is nonobstructive. Every hollow viscus is subject to at least two types of pathological condition, blockage of its lumen and inflammation of its wall, and the appendix is no exception. Obstruction to the lumen of this organ is comparable to intestinal obstruction in that it may be acute or chronic, partial or complete; yet the structure of the appendix favors the probability of acute complete obstruction in the majority of instances.

The factors which may be responsible for appendicular obstruction are many and varied. The intraluminal factors which may produce obstruction include fecaliths, fibrous strictures, foreign bodies, lymphoid hyperplasia, and carcinoid tumors. Impacted fecaliths comprise by far the largest percentage of etiological agents, 78 per cent in this group of cases (Fig. 1). Fibrous strictures probably account for the next largest group and often are associated with fecaliths. Fibrotic strictures are manifested by thickening and fibrosis of the submucosa, the same changes which are present as residua following acute or subacute inflammation (Fig. 2). Stricture often is missed owing to proximity to the cecum. Adhesions, secondary to inflammation in the appendix or adjacent organs, kinks, abnormalities of position, and peritoneal bands or membranes, comprise the extraluminal factors of obstruction.

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PATHOLOGICAL PICTURE

Gross examination alone usually provides sufficient evidence to classify a case of acute appendicitis as obstructive or nonobstructive. The circumstances surrounding the organ at the time of operation and its appearance *in situ*, together with a careful examination of the excised appendix, should furnish an etiological factor responsible for the pathological condition. Dilatation of the lumen and thinning of the walls are the two salient features that characterize an acutely obstructed appendix (Fig. 3). The luminal contents are always liquid and under pressure. Passive congestion, acute inflammation, and necrosis are absent in the earlier stages but they successively appear as the pathological process progresses. These changes are even more impressive when the site of obstruction lies distal to the base of the appendix, for then the changes in the appendix distal to the site of obstruction may be contrasted with the normal portion proximal to the site of obstruction, which has a lumen of normal caliber and walls of average thickness. The transition from normal to abnormal may be very abrupt. It is this feature particularly which favors the hypothesis of a primary mechanical etiological agent. Figure 4 shows graphically the anatomical picture at three different levels in the same appendix representing an advanced stage of acute appendicular obstruction.

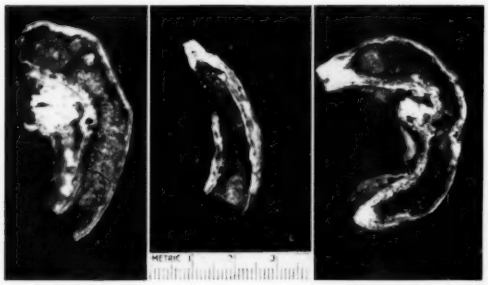

Fig. 1. Impaction of fecaliths in the proximal portions of 3 appendices, producing complete obstruction with distal dilatation of the lumina and thinning of the walls.

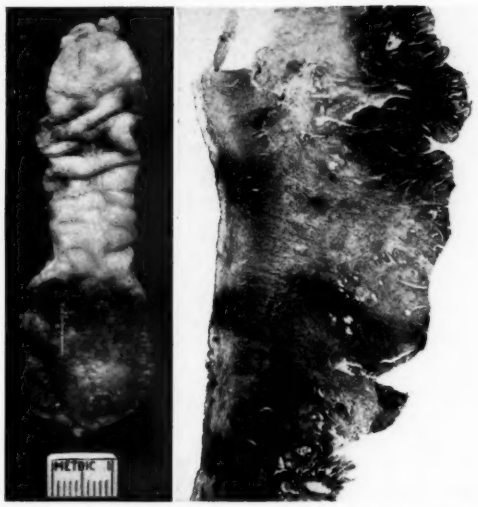

Fig. 2. a, left, An excised appendix which has been laid open to demonstrate better the abrupt demarcation of normal from abnormal at the site of an obstructing stricture. b, A longitudinal microscopic section through the region of stricture showing the intact normal mucosa proximal to the region of fibrous stricture and definite distal thinning of the wall and necrosis. ×8.

The increasing pressure of the intraluminal contents distal to the obstruction, the physiological characteristics of which have been described excellently by Van Zwalenburg (5), Sperling, Burget and co-workers, and Bowers quite naturally produces compression changes in the individual anatomical layers of the appendical wall. These changes are quite typical, and usually the microscopic examination of a single region is enough to establish a diagnosis (Fig. 5). Microscopically we find that the increasing intraluminal pressure has smoothed out the mucosal folds distal to the obstruction, has flattened the columnar cells that line the mucosal surface, has distorted and compressed the mucosal glands into a circumferential axis, has spread apart the individual glands so that there are fewer glands per high power field, has flattened the lymphoid aggregates, and has produced compression and thinning of the submucosa and muscularis.

In studying this series of obstructed appendices it was found that, from the standpoint of pathology, the cases could be classified in two major groups. The first group represents an early stage in which there is a definite obstructive mechanism with typical changes distal to the site of obstruction but no evidence of acute inflammatory infiltration (Fig. 6a). The second group represents the later stage of the process in which acute inflammatory infiltration is present distal to the site of obstruction (Fig. 6b). The only difference between the two groups is complicating infection with acute inflammation and eventual necrosis. One quite logically assumes, therefore, that the

Fig. 3. A longitudinal section of an obstructed appendix illustrating a proximal portion in which the lumen is of normal caliber and the walls are of average thickness and free from inflammation, an impacted fecalith with its typical laminated structure, and a distal portion in which there are the characteristic dilatation of the lumen and thinning of the walls. Distally there are passive congestion, acute inflammation, and foci of necrosis. The ease of rupture under these circumstances is evident. Note the abrupt transition from normal to abnormal at the site of obstruction. ×1½.

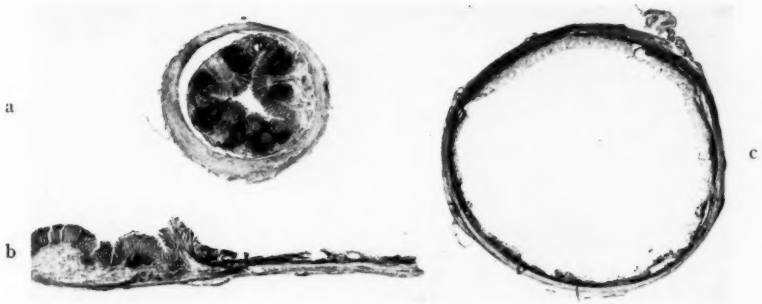

Fig. 4. a, A transverse section through an appendix proximal to the site of obstruction showing perfectly normal architecture free from inflammation and passive congestion. ×3.5. b, A longitudinal section at the site of obstruction by a fecalith in the same appendix. Note the abrupt demarcation of normal from abnormal. ×3.5. c, A transverse section distal to the obstruction illustrating a markedly dilated lumen and thinned walls which are necrotic, congested and infiltrated with cells characteristic of acute inflammation. ×3.5.

second group represents only a later stage in the pathological process. When obstruction becomes complete, the pathological process becomes progressive. The microscopic picture of the organ does not represent a complete description of the disease but only a stage of the disease at the time of appendectomy.

In contradistinction to the preceding picture of an acutely obstructed appendix, the nonobstructive type of acute appendicitis does not show any distention of the lumen or thinning of the walls (Fig. 7). Rather there are thickening and bogginess of the walls due to edema and inflammatory infiltration. The muscularis is thick and edematous and its fibers are separated by inflammatory exudate. The mucosal architecture shows none of the aforementioned compressive changes.

CLINICAL PICTURE

In studying the clinical history and findings for the entire series it was apparent that the most characteristic symptom in the majority of cases of appendicular obstruction was a colicky type of abdominal pain. The sudden onset of the pain and its intermittence were also suggestive. The intermittent character of a pain is particularly suggestive, for if the initial pain were due to simple infection, the pain would not subside so frequently after a few minutes or hours while the infection continues to extend. Systemic signs of fever, rapid pulse, and leucocytosis may be absent in acute appendicular obstruction even when the pathological picture appears dangerously acute. Yet in the advanced or late stage of the disease they are usually present. This is at variance with the syndrome described by Wilkie and by Boyce and McFetridge, whose emphasis on the lack of systemic manifestations has led many to suppose that this is characteristic of acute appendicular obstruction. One can say merely that such constitutional signs are usually absent only in the earlier stages and are usually present in the advanced stages. Vomiting is not a characteristic sign of appendical obstruction; it is as frequently absent as it is present. In all cases some degree of abdominal tenderness was present. The differentiation between acute obstructive and nonobstructive types of appendicitis certainly cannot always be made clinically, but in a very large per-

Fig. 5. Transverse section of an appendix distal to the obstruction, illustrating an early phase of acute obstruction with its dilated lumen, thin walls, typical compression changes, and no evidence of acute inflammation. ×7.

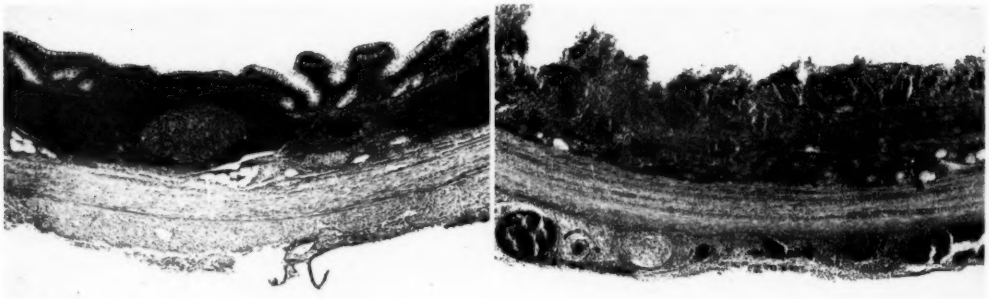

Fig. 6. a, left, The compression changes in anatomic layers of the appendical wall resulting from increasing intraluminal pressure in the early stage of appendicular obstruction. Smoothing out of the mucosal folds, flattening of the surface epithelium, compression of the mucosal glands into a circumferential axis, spreading apart of the individual glands, flattening of the lymphoid aggregates, and compression of the submucosa and muscularis are all evident. ×25. b, A transverse distal section in an advanced stage of acute appendicular obstruction showing the necrosis, acute passive congestion, hemorrhage, thrombosis and acute inflammatory exudate. ×25.

centage of cases the symptoms are suggestive enough to make one strongly suspect that the obstructive variety is present.

REPORT OF CASES

The following reports of cases are illustrative. The first two are typical of the group of early cases of acute appendicular obstruction before bacterial invasion and inflammation complicate the picture. In Case 1 at operation with the appendix *in situ* the impression of the surgeon was that of an acutely obstructed appendix. Microscopic examination by the pathologist did not reveal any acute inflammation and the condition was labeled as only chronic, thereby minimizing to the clinician the potential danger of the situation. Given a few more hours in which the obstructive factor could produce more passive congestion, distention, and pressure necrosis, the appendical walls would be permeated with cells characteristic of acute inflammation and gangrene and perforation would be approaching.

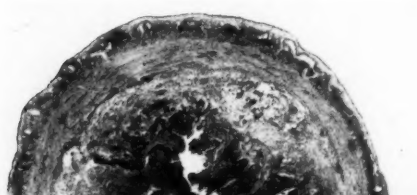

Fig. 7. Transverse section through an appendix that is the site of a nonobstructive type of acute appendicitis, illustrating the typical thickening and bogginess of the walls which are infiltrated with acute inflammatory exudate. The mucosa has become necrotic. ×7.

Case 1. A man, aged 25 years, experienced the abrupt onset of intermittent colicky abdominal pain 48 hours before operation was performed. There were several intervals of complete freedom from pain. During the last few hours before operation, the pain centered in the right lower quadrant of the abdomen and became more steady. The temperature, pulse rate, and number of leucocytes were within normal range. At operation the appendix was found to be definitely obstructed by a fecalith and distal to the site of obstruction to be distended with liquid fecal contents under pressure. To the surgeon the condition represented acute appendicular obstruction. Microscopically the distal walls of the appendix showed the characteristic changes due to compression, but no cells characteristic of acute inflammation were found.

Case 2. A girl, aged 18 years, presented an 18 hour history beginning with an abrupt onset of intermittent cramp-like pains in the lower part of the abdomen with particular extension into the right lower quadrant of the abdomen. Nausea became apparent in a few hours but there was no vomiting. The patient's temperature was 98.5 degrees F.; pulse, 85; leucocytes numbered 11,500 per cubic millimeter of blood; 79 per cent were neutrophils. Examination disclosed some tenderness in the right lower quadrant of the abdomen. Appendectomy revealed several fecaliths which were definitely obstructing the distal two-thirds of the organ. Microscopic examination showed proximal transverse sections to be normal. Distal sections revealed a dilated lumen, thinned walls with typical compression changes, and moderate passive congestion and hemorrhages. There was no evidence of acute inflammatory infiltration. Because of this absence of inflammation the pathologist classified the condition merely as chronic appendicitis.

The next case represents the advanced stage of acute appendicular obstruction wherein bacterial invasion with an acute inflammatory tissue response has been superimposed on the primary picture of acute mechanical obstruction.

CASE 3. A woman, aged 30 years, presented an 18 hour history beginning with an abrupt onset of colicky abdominal pain located always around the umbilicus. The pain was intermittent with several intervals of complete freedom from pain. Temperature, pulse, and number of leucocytes were all elevated above normal limits. Examination revealed slight tenderness in the right lower quadrant of the abdomen and no rigidity. The appendix was found to be acutely inflamed and obstructed in its distal third which was dilated considerably by liquid contents under pressure. Examination disclosed an obstructing stricture in the middle third with a fecalith impacted behind it. Microscopically the transverse sections proximal to the obstruction were normal and intact, whereas distally the walls were compressed and thin, necrotic, and permeated with cells characteristic of acute inflammation and residua of hemorrhage (Fig. 4).

OBSERVATIONS

It is apparent, therefore, that appendicular obstruction presents two phases in its development. The first or early phase is that of primary simple obstruction characterized by symptoms suggestive of obstruction and usually with no elevation of temperature, pulse rate, or number of leucocytes. In some instances the obstruction may be partial and occasionally may be relieved spontaneously. The patient may go on with apparently insignificant symptoms for several days before obstruction becomes complete. The rapidity with which the clinical picture develops depends on the completeness and suddenness of the obstruction. The pathological picture corresponds to the clinical manifestations. In the early stage one would not expect and does not find a microscopic picture of an acute process with inflammatory exudate. The late phase begins when the supply of blood to the wall is impaired, owing to increasing intraluminal pressure; then, bacteria begin to invade the wall with resultant acute inflammatory infiltration. Clinically, an elevated temperature and leucocytosis develop. At this stage a diagnosis of acute appendicitis is made easily.

The didactic classification by pathologists of appendices into separate categories, such as "acute suppurative," "simple catarrhal," and "gangrenous," simply portrays the microscopic picture of the organ at the time the surgeon intervened. It must be emphasized again that it is not a description of the disease but of only a stage of the disease at the time of appendectomy. The disease within several hours might have regressed or might have merged rapidly into a more acute picture, and in the latter instance the microscopic appearance would be very different. This is particularly the case in appendicular obstruction. It is usually a progressive condition, and if the appendix examined by the pathologist shows evidence of only minor changes, it is no criterion that the removal of the appendix was not a vital necessity, for a few more hours might have sufficed to change an innocent appearing structure into a gangrenous one. It is only the early decision of the surgeon based on the clinical picture and his knowledge of possibilities which prevents the change. The speed with which such changes take place is dependent on the completeness of the obstruction and not so much on the duration of the condition.

It should be borne in mind that the microscopic appearance of the appendix is not the sole criterion of the symptomatology. The circumstances surrounding the organ and its appearance *in situ* are often the explanation of the clinical signs and symptoms and the determining factor of what might have happened in a short time. Once these are disturbed one of the most important parts of the story is lost and important facts are concealed from the pathologist, if they are not recorded. The surgeon and the pathologist must co-operate in the study of the etiological factors lest their true significance be lost.

In the obstructive variety of appendicitis the pathological picture is usually progressive. Tension may mount extremely rapidly within the lumen and gangrene and rupture may occur in an exceedingly short time. Time is a much more important factor in the obstructive variety. The average of my advanced series is 24 hours and in most of these cases there were regions of gangrene. Even at the end of 6 hours one specimen showed definite necrosis. Interference with blood supply and increasing intraluminal pressure are the causative factors. If the history is suggestive of acute appendicular obstruction, surgical intervention is urgent. It is reasonable to suppose that rupture of an appendix whose infectious factor has only a short duration is much more serious than that of an appendix which is the seat of a nonobstructive appendicitis in which the infection begins at the onset of the illness and extends over a much longer time. In the latter instance the surrounding peritoneal cavity and omentum have a better opportunity of preparing for the rupture. The fact that an acutely obstructed gangrenous appendix usually lies free in the peritoneal cavity with little attempt by the omentum to surround it has been noted by many surgeons.

The foregoing discussion emphasizes the importance of realizing what occurs in acute appendicular obstruction and the value of distinguishing, when possible, between primary obstruction and primary inflammation. It explains the cases in which the patient remains under observation during several days of intermittent symptoms with normal temperature, pulse, and leucocyte count, and then acute, rapidly progressive appendicitis suddenly develops. The value of clinical suspicion lies in the early surgical intervention that it encourages.

One can appreciate, therefore, the importance of recognizing or suspecting this type of appendix in its early stage. If one waits until the signs of fever and leucocytosis appear, time is at a premium and immediate operation is urgent. It is like waiting for a strangulated hernia to become gangrenous.

SUMMARY

An attempt has been made to correlate the clinical and pathological picture seen in 70 cases in which operation was performed for an obstructive type of appendicitis. Gross examination of the organ usually provides sufficient evidence to classify a case of acute appendicitis as obstructive or nonobstructive. A characteristic microscopic picture confirms such evidence. Appendicular obstruction presents two phases in its development: the first, or early, phase is that of primary simple obstruction; the second, or late, phase begins when the supply of blood to the wall is impaired and bacteria invade the wall with resultant acute inflammation. The significance of the microscopic appearance of the early stage has been emphasized. Inasmuch as obstruction is one of the frequent factors in the production of appendicitis, the importance of its early recognition and treatment has been stressed.

REFERENCES

1. Bowers, W. F. Arch. Surg., 1939, 39: 362–422.
2. Boyce, F. F., and McFetridge, Elizabeth, M. Internat. S. Digest., 1936, 22: 195–213.
3. Burget, G. E., Martzloff, Karl, Suckow, George, and Thornton, R. C. B. Arch. Surg., 1930, 21: 829–837.
4. Sperling, Louis. Arch. Surg., 1938, 36: 778–815.
5. Van Zwalenburg, C. J. Am. M. Ass., 1904, 42: 820–827.
6. Idem. Ann. Surg., 1905, 41: 437–450.
7. Wangensteen, O. H., and Bowers, W. F. Arch. Surg., 1937, 34: 496–526.
8. Wilkie, D. P. D. Brit. M. J., 1914, 2: 959–962.

THE SEX HORMONE EXCRETION OF ADULT FEMALE AND PREGNANT MONKEYS

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ALTHOUGH the monkey has been extensively employed for studies in reproduction, little is known of the normal metabolism of the steroid sex hormones in this species. Some work has been reported on the levels of the estrogenic and androgenic substances in the urine of the adult male and immature monkeys (5, 6, 7). This communication deals primarily with determinations of urinary estrogens and androgens in adult female and pregnant monkeys.

It has been found that if the fetus is removed some time between the seventieth and one hundred and fifty-seventh day of gestation, the placenta remains *in situ* and is expelled at the time when a living baby might have been delivered. From the gross appearances of generalized edema and intensification of the sex skin color, it appears that the animals maintain the habitus of pregnancy in spite of the absence of the fetus (11). We have studied the sex hormone excretion under these conditions to learn more of the endocrine state. We have included also in this report the study of a pregnant monkey in which the fetus was removed and the animal simultaneously bilaterally ovariectomized.

The results in this animal bring some direct evidence upon the rôle of the placenta and the adrenal cortex in the production of the steroid sex hormones.

ANIMALS AND METHODS

Ten adult female rhesus monkeys (Macaca mulatta) were used in these experiments. For the studies on the concentrations of sex hormones in normal females, 6 animals were employed from which ten 3 day samples were quantitatively collected. A total of 4 normal pregnancies in animals A, B, and C were investigated, and at intervals of 2 weeks samples of urine were collected over a period of 3 days during each gestation. In addition, 4 other pregnancies were similarly studied in animals D, E, F, and G, in all of which, however, some form of operative intervention had been used. In 3 animals, D, E, and F, the fetus only was removed by abdominal hysterotomy at different intervals before term, but in animal G the ovaries also were excised when the fetus was taken out upon the seventieth day of gestation.

All urines were preserved with toluene during the periods of collection. The urinary concentrates were separated into the estrogenic and androgenic fractions, respectively, as previously described (8). The androgens were assayed by means of the day old chick method (4), at least 10 animals being used for each assay. All values for androgenic substances are expressed as international units (I. U.), which is that activity evoked by 0.1 milligram of androsterone. The estrogenic activity was assayed by means of the vaginal smear reaction in the spayed adult female mouse and all values for estrogenic material are expressed as international units (I.U.), which is that activity evoked by 0.1 microgram of estrone.

RESULTS

The urinary excretion of androgenic and estrogenic substances by the adult female monkey is presented in Table I. No attempt was made to study the daily fluctuations of these androgens and estrogens, but rather to delimit the urinary concentrations of these substances. A total of 10 samples, each representing a composite of 3 days' urine, were collected from 6 different animals. The urinary content of estrogenic substances varied from 7 to 12 international units per 24 hours, while the concentration of androgenic substances varied from 1.2 to 3.6 international units per 24 hours with an average value of 2.2 international units.

Figure 1 illustrates the variations in the excretion of androgens and estrogens during 4 normal pregnancies in 3 monkeys (A, B, C). Definite increases in estrogenic substances are detectable between the thirtieth and sixtieth day of gestation. The highest value for estrogenic substances was found in monkey C at 114 days after conception when a value of 460 international units per day was determined. This represents a 46 fold increase above the nonpregnant levels for estro-

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TABLE I.—THE SEX HORMONE EXCRETION IN THE URINE OF THE ADULT FEMALE MONKEY

Monkey	Days of cycle	Androgenic substances I.U./24 hrs.	Estrogenic substances I.U./24 hrs.
J	7, 8, 9	3.6	7
E	9, 10, 11	2.0	<10
C	11, 12, 13	1.8	9
H	11, 12, 13	2.0	<10
H	14, 15, 16	2.5	8
E	17, 18, 19	1.5	8
D	24, 25, 26	1.9	<10
J	25, 26, 27	2.5	12
C	27, 28, 29	2.7	<15
I	34, 35, 36	1.2	11

genic substances. Within 2 weeks after parturition the quantity of estrogens returned to normal nonpregnant levels.

Within the first half of the gestation period the androgenic substances showed a considerable increase over the normal nonpregnant values. In 3 of the 4 pregnancies studied values of androgenic material above 6 international units per day were recorded in comparison with the average value of 2.2 international units per day for the nonpregnant levels. In the fourth pregnancy the values for androgens were not as great as in the other 3 pregnancies, but still one value of 4.7 international units per day was recorded in contrast to the maximum value of 3.6 international units per day recorded for the normal adult females.

Within the first postpartum month the androgenic titer remained at a high level. Thus, in the case of monkey A between the thirteenth and sixteenth day the titer of androgenic substance was 4.0 international units, and between the twenty-seventh to thirtieth day, 5.0 international units. However, the concentration of androgenic substances returns to normal limits during the second postpartum month, as evidenced by the fact that in monkey A a value of 1.0 international units per day was found by the forty-eighth day after delivery.

The influence of the removal of the fetus on the urinary excretion of the sex hormones is presented in Figure 2. Three such cases were studied which included monkeys D, E, and F, from which the fetuses were removed by abdominal hysterotomy at the seventy-first, one hundred and forty-first, and one hundred and fifty-seventh day, respectively. The placentas were retained and finally expelled near the expected date of parturition.

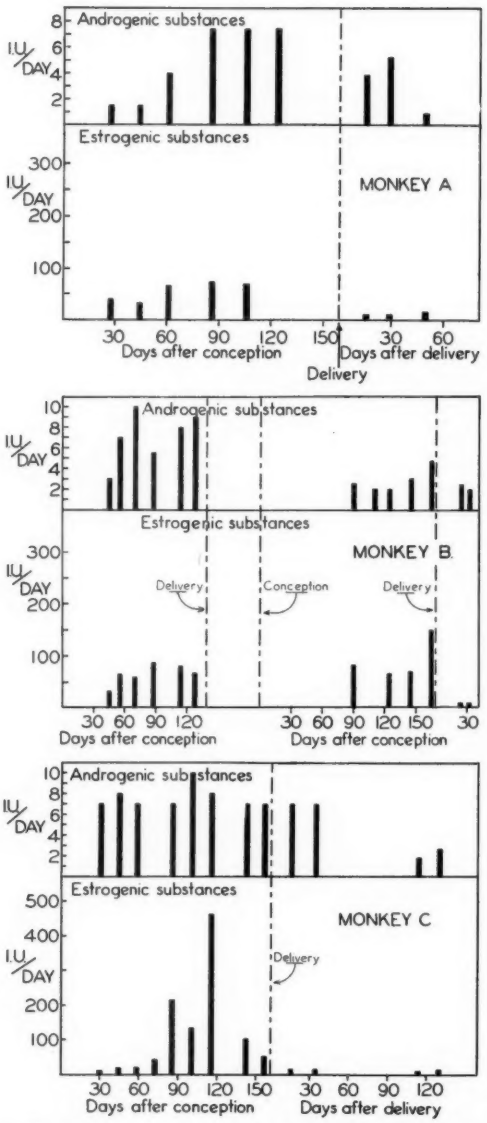

Fig. 1. The urinary excretion of estrogenic and androgenic substances by the pregnant monkey.

The removal of the fetuses caused no significant change in the urinary excretion of the sex hormones.

Figure 3 presents a study of the excretion of estrogens and androgens during a pregnancy in

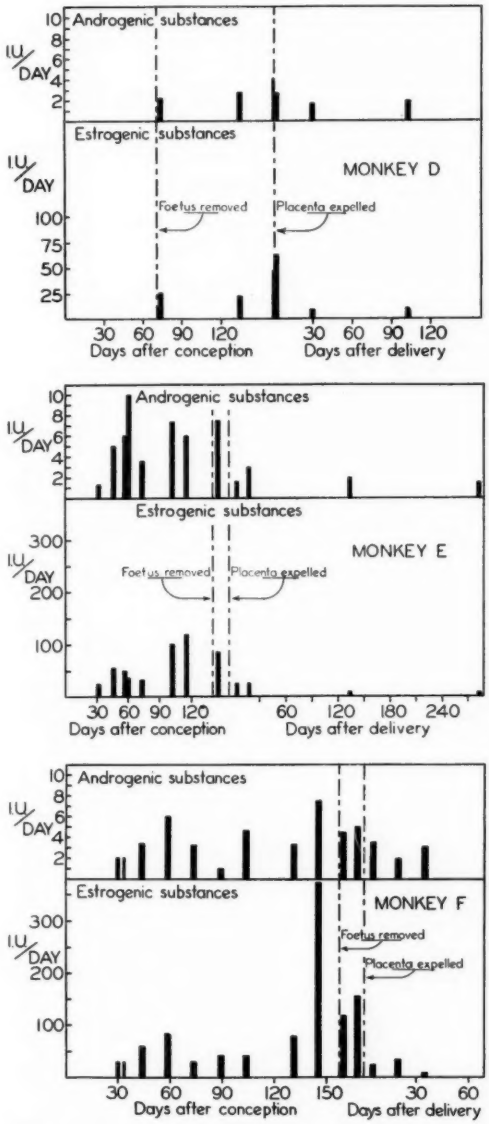

Fig. 2. The urinary excretion of estrogenic and androgenic substances by the pregnant monkey when the fetus was removed before term.

which animal G had been subjected to ovariectomy on the seventy-third day at the time of the removal of the fetus. The placenta was retained until the one hundred and fifty-third day, at

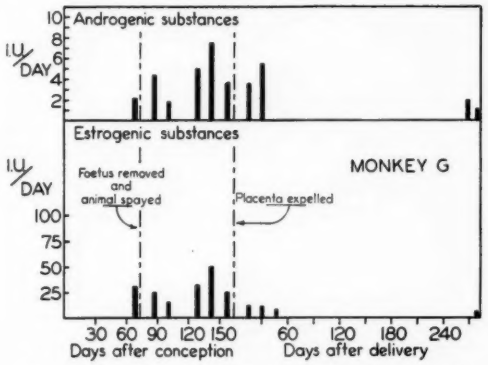

Fig. 3. The urinary excretion of estrogenic and androgenic substances by the pregnant monkey after removal of both ovaries and the fetus.

which time it was expelled. Throughout the period of placental retention the quantity of urinary estrogens was maintained well above the nonpregnant excretion figures of 7 to 12 international units, ranging from 15 to 50 international units per 24 hours. The androgen excretion during this same interval ranged from 1.8 to 7.5 international units per day with an average of 4.6 international units, and was definitely above the control values of 1.2 and 2.0 international units found about 6 months after the expulsion of the placenta. As in the case of normal pregnancy, the high titer of androgenic substances was maintained during the first postpartum month.

EVALUATION OF STUDY

The comparative urinary excretion of estrogenic and of androgenic substances in the monkey have been summarized in Table II. The immature animal excretes very little, if any, substance possessing estrogenic or androgenic activity. Both the adult male and female monkey, however, excrete measurable amounts of active substances. With respect to the estrogens, the female of this species seems to excrete considerably more than the male. The urinary excretion of androgenic substances is of interest since the quantity of these materials is of the same magnitude in both males and females, the average being 2.6 and 2.2 international units per day, respectively.

During pregnancy in the monkey we find increased concentrations as both estrogenic and androgenic substances in the urine; this, in the case of estrogens, may be as great as 46 times that of the normal female. The increase in estrogenic substances during pregnancy agrees with results found for the human (1).

TABLE II.—THE COMPARATIVE URINARY EXCRETION OF ESTROGENIC AND ANDROGENIC SUBSTANCES IN MONKEYS

References to literature	Sex of animals	Status of animals	Number of animals	Number of days' urine collected	Androgenic substances I.U./Day (range)	Extrogenic substances I.U./Day (range)
(5), (6)	♀	Immature	2	20	<0.05	<0.8
(5), (6)	♂	Immature	2	20	<0.05	<0.8
This report	♀	Adult	6	30	2.2	(7–12)
This report	♀	Pregnant	3	78	5.9* 1.5–10.0	88 10–460
(7)	♂	Adult	3	46	2.6 1.0–4.7	1 1.1–2.5

*This is a minimum value since 10 of the 26 assays were minimum assays due to lack of extract.

In keeping with previous observations in the monkey that the removal of the fetus does not interfere with the general status of pregnancy (11), we find that no significant change occurs in the excretion of estrogenic substances. When the pregnant animal was ovariectomized at the same time that the fetus was removed, the concentration of urinary estrogens was maintained well above the nonpregnant levels. The greater part of these estrogens was probably of placental origin because it was further noted that when the placenta was expelled the estrogen excretion definitely decreased. The source of the residual estrogens, that is, in the absence of both placenta and ovaries, was probably the adrenal cortex. The fact that the adrenal cortex can produce estrogens is well established, especially since estrone has been isolated from this source (2). Thus, it is apparent that three sources, the ovaries, the placenta, and the adrenal cortex, contribute to the production of estrogens during the period of pregnancy.

The presence of increased quantities of androgenic substances during pregnancy in the monkey is of considerable interest. The fact that this increase persists for about a month after the expulsion of the placenta in animals with or without ovaries indicates that the increased androgen production during pregnancy is due to the adrenal cortex. Hypertrophy of the adrenal cortex during pregnancy has been described in the human (10) and in the guinea pig (9). The rhesus monkey is the first animal in which an increased quantity of *urinary androgens* has been demonstrated during pregnancy. Studies in humans have not indicated similar increases (3).

SUMMARY

Studies have been made on the sex hormone excretion of (a) adult female monkeys, (b) pregnant monkeys, (c) pregnant monkeys from which the fetuses had been removed by abdominal hysterotomy, and (d) a pregnant monkey which was ovariectomized early in gestation when the fetus was removed.

Increases in both estrogenic and androgenic substances in the urine of pregnant monkeys were observed. Following parturition, or expulsion of the placenta alone, the estrogens immediately decrease to the nonpregnant values while the high concentration of androgens persists for approximately 1 month post partum. Direct proof is presented that the estrogens may be produced in the ovary, placenta, and adrenal cortex. The results indicate that the greatest part of the androgens produced during pregnancy are derived from the adrenal cortex.

The rhesus monkey is the first animal in which an increase in the urinary androgens has been demonstrated in pregnancy. The excretion of estrogenic and of androgenic substances is compared in immature, adult, and pregnant monkeys.

REFERENCES

1. Aschheim, S., and Zondek, B. Klin. Wchnschr., 1927, 6: 1322.
2. Beall, D. J. Endocrinology, 1940, 2: 81.
3. Dingemanse, E., Borchardt, H., and Laqueur, E. Bio-Chem. J., 1937, 31: 500.
4. Dorfman, R. I., and Greulich, W. W. Yale J. Biol. Med., 1937, 10: 79.
5. Dorfman, R. I., and Hamilton, J. B. Endocrinology, 1939, 25: 28.
6. Ibid. 1939, 25: 33.
7. Dorfman, R. I., and Van Wagenen, G. Proc. Soc. Exper. Biol. & Med., 1938, 39: 35.
8. Gallagher, T. F., Peterson, D. H., Dorfman, R. I., Kenyon, A. T., and Koch, F. C. J. Clin. Invest., 1937, 16: 695.
9. Hewitt, W. F., and Van Liere, E. J. Endocrinology, 1941, 28: 62.
10. Stoerk, O., and Haberer, H. v. Arch. f. mikr. Anat., 1908, 72: 481.
11. Van Wagenen, G., and Newton, W. H. Am. J. Physiol., 1940, 129: 485.

CECOSTOMY

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Boston, Massachusetts

THE importance of cecostomy in dealing with malignant lesions of the left colon has been recognized for many years. Recent advocates of this operation are Whipple and Rankin. Early procedures of this type were for relief of complete obstruction, and this is still the only use made of it by many surgeons, either as a palliative operation or as a preliminary procedure to resection. When obstruction of a marked degree does not exist, cecostomy is usually practiced as a complementary step in primary resection. On the whole, the application of these principles has given good results, particularly when the cases have been well selected and in the hands of certain surgeons. We believe our results (1) indicate that preliminary cecostomy, in all lesions of the left colon and in some cases of carcinoma of the rectum followed by resection at a later stage, will result in a lower mortality rate than will cecostomy coincident with primary resection.

From the East Surgical Service, Massachusetts General Hospital and the Department of Surgery, Harvard Medical School.

Our experience has been greater with resection and anastomosis of the bowel than with any of the various exteriorization or obstructive resection methods which are frequently used elsewhere. This probably explains our improved results in cases in which a preliminary cecostomy is performed 7 to 10 days prior to resection of the lesion, since leakage, peritonitis, or wound sepsis rarely follow resection and suture under these circumstances.

The earlier cecostomies consisted of exteriorization of the cecum and, in many instances, fixation of the bowel to the abdominal wall. This method is still widely used and when the vent is to be permanent, it is the procedure of choice. Many

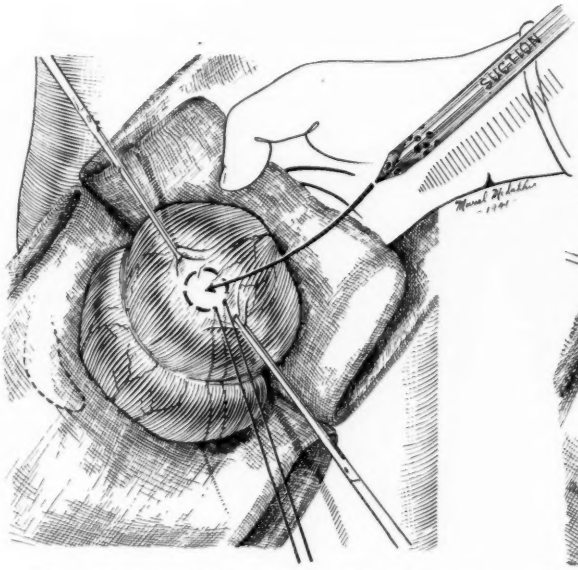

Fig. 1. The distended cecum has been delivered through a McBurney incision. The wound edges are carefully protected with gauze. A pursestring of No. 0 plain catgut has been placed, and suction trochar about to be introduced.

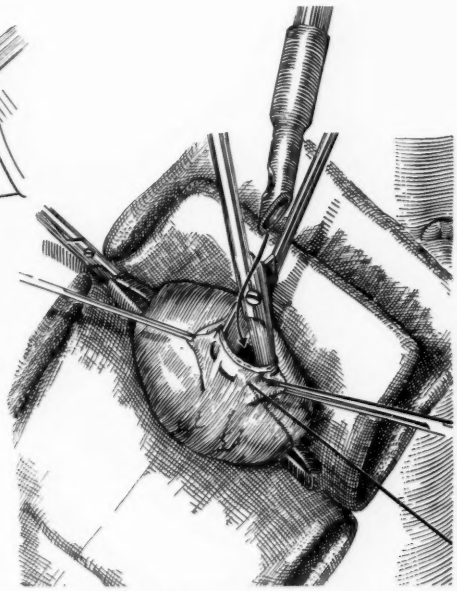

Fig. 2. The cecum has been decompressed and a rubber covered clamp applied. The cecum has been opened. As the scissors are spread, the rubber covering the right angle glass tube is inserted.

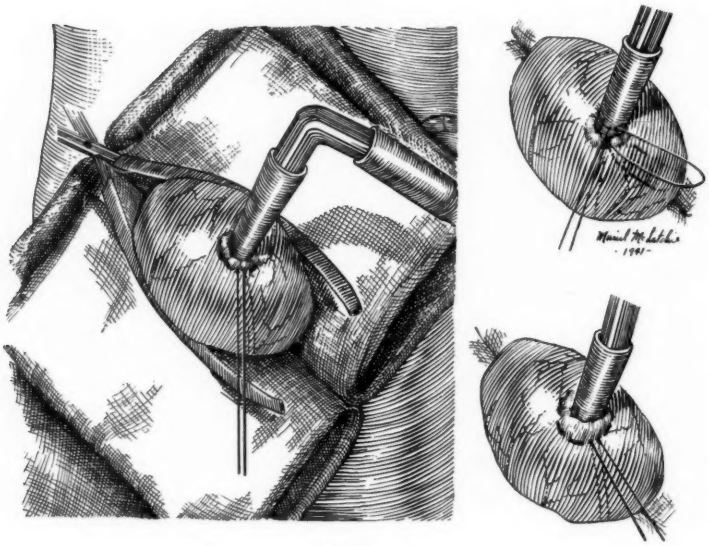

Fig. 3.

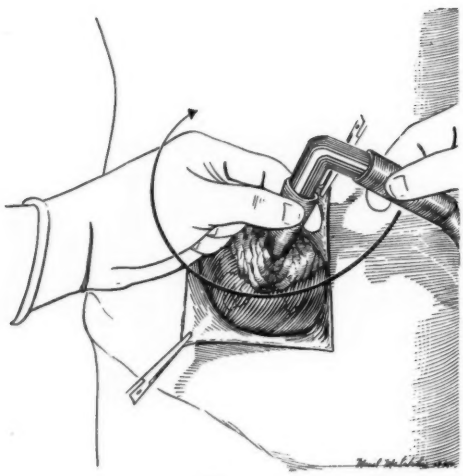

Fig. 4.

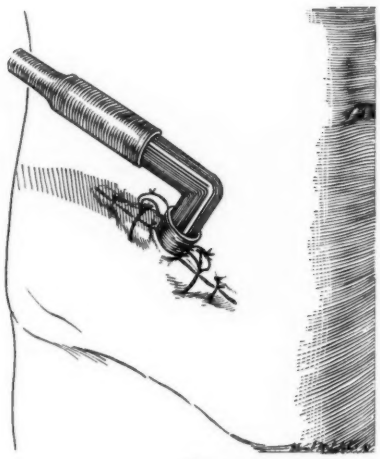

Fig. 5.

Fig. 3. The pursestring is tightened and the same suture passed through the tube and tied. A second inverting pursestring of No. 0 chromic catgut is then placed.

Fig. 4. A third pursestring of No. 0 chromic catgut has been inserted. The tip of the omentum has been sutured about the tube. The operator then holds the rubber tube firmly and rotates the glass tube about until it reaches its final position.

Fig. 5. The final appearance after wound closure. One through-and-through suture is tied on either side of the tube, and then passed through the rubber as an additional protection to maintain the position of the tube.

suggestions have been made for temporary cecostomy by the use of glass or rubber tubes of varying designs. When complementary cecostomy is to be established, the Pezzer catheter type of Hendon or Nason, with Nicholson's modification, is satisfactory. This method, however, has some disadvantages as a preliminary procedure. The glass tube method of Paul, or Mixter's (4, 7) modification of it, is also unsatisfactory since leakage about the tube occurs so soon after operation that control of the fecal current is quickly lost. Gibson advocated the use of a large rubber tube for cecal drainage in ulcerative colitis, and Jones used this method extensively in cecostomy as a preliminary and coincident procedure for carcinoma of the left colon.

We have found that a short segment of large rubber tubing, fitted on a right angle glass tube, gives the most uniform result in temporary cecostomy of any of the methods so far used. The rubber tubing holds well in the bowel wall and can be so placed by 3 pursestring sutures of catgut that the serous surfaces of the vent come in contact when the tube is removed, thus enhancing a spontaneous closure of the opening. The glass right angle tube, going through some or all of the abdominal wall, prevents kinking of the tubing, which is the chief disadvantage of any all rubber apparatus that we have tried. This rigid segment also makes it easier to fix the tube securely to the abdominal wall in such a manner that change of position can be accomplished by the patient with as little discomfort as possible.

The bowel can usually be well cleansed by this procedure. After 24 hours, dilute magnesium sulphate can be instilled directly into the colon several times daily. Often salt solution can be irrigated through the lesion from the rectum to the cecostomy or vice versa. Occasionally, the bowel cannot be as thoroughly cleansed by this method as by complete colostomy and, for this reason, in large obstructed and grossly infected growths, one may do well to use transverse colostomy or Devine's modification of it either instead of, or, at times, following cecostomy before resection of the involved bowel is undertaken. It is well to bear in mind that the contents of the colon should be as dry as it is possible to make them at the time of resection, since liquid feces in small quantities are far more dangerous to the peritoneal cavity than firm scybala. For this reason, no irrigations of the colon should be made for 24 to 36 hours before resection, and, during this interval, paregoric in dram doses should be given every 4 hours.

The disadvantages of temporary cecostomy are:

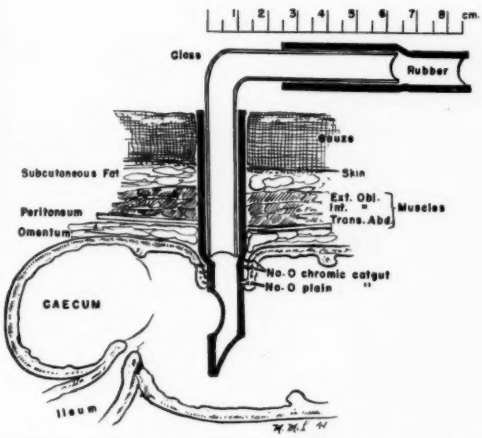

Fig. 6. A cross section of the tube in its final position.

(1) Wound sepsis invariably occurs to some extent and can be controlled by removal of sutures and warm boric acid solution compresses. Serious infection rarely, if ever, occurs and has caused no deaths in our series. (2) Hernia in scar is common, due to the long maintenance of the foreign body through the abdominal wall. Since it is necessary to keep the cecostomy open until the anastomosis is functioning well, this means that the tube remains in place for from 2 to 3 weeks. Usually these hernias cause little trouble and rarely do they warrant repair. (3) Failure of the cecostomy to close spontaneously occurs occasionally. It is wise to wait a minimum of 3 months and often complete cessation of drainage will take place long after that. Only when the mucous surface of the opening presents above the skin edges can one be sure that spontaneous closure will not take place, and this situation is rarely met if the procedure is carried out as we have described it. On the whole, the complications are a small price to pay for the numerous advantages of the procedure.

Our experience at the Massachusetts General Hospital with cecostomy, performed for malignant disease in a consecutive series of 250 cases ending January 1, 1941, is given in Table I.

TABLE I.

Method

	Cases
Mixter's glass tube	22
Witzel	23
Pezzer catheter or exteriorization of cecum	11
Modified Gibson (as described by us)	194
Total	250

There were 32 deaths in this series, 21 of which were due to perforated lesions or carcinomatosis. Eleven deaths were attributable to the cecostomy itself. In some instances, the fatality was due to an error in technique, in others to cardiac or pulmonary lesions.

Surgical closure of the cecostomy was necessary in 17 instances. There were 14 known recorded hernias in scar.

REFERENCES

1. Allen, A. W., and Welch, C. E. Am. J. Surg., 1939, 46: 171–180.
2. Devine, H. Surgery, 1938, 3: 165.
3. Gibson, C. L. Boston M. & S. J., 1902, 147: 341.
4. Gould, Alfred H. The Technic of Operations upon the Intestines and Stomach. Philadelphia: W. B. Saunders Co., 1906.
5. Hendon, G. A. Ann. Surg., 1931, 94: 156.
6. Jones, D. F. Tr. Am. Surg. Ass., 1931, 49: 308.
7. Mixter, S. J. Boston M. & S. J., 1895, 132: 206.
8. Nason, L. H. Am. J. Surg., 1936, 34: 292.
9. Nicholson, W. P., Jr. Ann. Surg., 1937, 105: 878–880.
10. Paul, F. T. Brit. M. J., 1895, 1: 1139.
11. Rankin, Fred W. Tr. Am. Surg. Ass., 1939, 57: 44–52.
12. Whipple, Allen O. J. Am. M. Ass., 1931, 97: 1962–1964.

EDITORIALS

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OCTOBER, 1941

BRAZILIAN OPHTHALMOLOGY

I HAVE been requested by the Editorial Board of SURGERY, GYNECOLOGY AND OBSTETRICS to record my impressions of the practice of ophthalmology in Brazil after attending the IV biennial Brazilian Congress of Ophthalmology, held in Rio de Janeiro the last week of June of this year. Previously to that time, I knew one Brazilian ophthalmologist intimately and several others only very casually. I knew nothing of the methods or character of practice there. But I have come away with a very favorable impression.

The men from forty years of age upward were for the most part trained either directly or indirectly in the ophthalmic clinics of Central Europe, particularly Germany and Austria. But there is definite evidence that they thought for themselves and do not adhere slavishly to the fetishes of the individual clinic in which they spent the majority of their time. A few have supplemented their knowledge by visits of varying length of time in the clinical centers of the United States. French and Italian influence, which formerly was in the ascendency in Brazilian ophthalmic circles, has now entirely disappeared. The men of that generation are fairly conversant with the work being done in the United States, although the knowledge has been gained from the literature and seldom from personal contact.

Nearly all of the younger men have been trained in Brazil by serving as junior assistants in the university eye clinics of which there are eleven. The same disadvantage exists there as did until recently in the United States, namely lack of formalized instruction particularly in the basic sciences of ophthalmology. The instruction is mainly clinical and somewhat of the preceptorship type. However, instructional courses are beginning to make their appearance and without doubt, the adolescent period from which American ophthalmology is just emerging, will be considerably shortened in Brazil. In the tropics, things mature rapidly.

There is a much sharper differentiation between charity and private practice than we are accustomed to. Hospitals, as we know them, partly charitable, partly half-pay, and partly private, do not exist there. A hospital in Brazil is always a charitable institution regardless of whether it is supported by the national or state government, by religious orders, or by private agencies. Private patients are operated upon in sanatoria or what corresponds to the British nursing home. Some ophthalmologists with large practices maintain small hospitals of their own, of 2 to 15 beds, in conjunction with their consulting offices. The Army and Navy hospitals are excellent.

The ophthalmic equipment in the private offices and in the public institutions is good.

The older instruments were almost invariably German, but they are being replaced rapidly by modern American equipment. None of the optical instruments or glass is of local make.

The treatment of eye, ear, nose, and throat conditions is done by the same man only in the smaller communities, while in cities of from 35,000 upward, the men confine themselves to one or the other specialty. Long working hours are the rule. Usually the men work in the clinics or the hospitals in the morning and conduct their private practice in the afternoon from two to seven. Dinner is always at a late hour, and there are practically no evening hours of consultation. Men on duty in the Army and Navy hospitals are permitted to conduct private practice in the afternoons, as are many of the public health officers.

The individual practice of ophthalmology in Brazil is much as it is in the United States. We use the same instruments of diagnosis, the same nomenclature, and the same surgical procedures. Therapeutic measures vary but slightly. Refraction is more apt to be performed under mydriasis in the United States, while in Brazil, refractometers, either Bausch or Zeiss, are much favored. Anomalies of the ocular muscles have been more or less neglected in the past, but are now receiving greater recognition, due mainly to British influence. Some orthoptic training is being instituted, again principally of English origin. In certain parts of Brazil, tropical diseases bring ophthalmic problems that we do not have to cope with; but on the whole, individual ophthalmology in Brazil is on the same plane as in the United States.

Graduate training in ophthalmology is inadequate in Brazil, due principally to a lack of organization. The Congress that was just held, voted to establish a Brazilian Board of Ophthalmology, patterned much along the lines of the American Board of Ophthalmology. That important decision will open the gate to ophthalmic organization, and because they can and will borrow from the experience of the American Board, progress will be more rapid in Brazil than it has been in the United States. There is a full realization there that ophthalmology for the future will be centered in the Americas, and the Brazilians are preparing to do their part. HARRY S. GRADLE

DIAGNOSIS, OPERABILITY OF INTRATHORACIC NEOPLASMS

INTRATHORACIC neoplasms usually present first symptoms, physical signs, and occasionally even roentgenological shadows that strongly suggest some commonplace thoracic disease. The great majority of thoracic neoplasm patients are first diagnosed as having a simple cold, influenza, pneumonia or delayed resolution of pneumonia, tuberculosis, asthma, pleurisy with effusion, chronic bronchitis, bronchiectasis, pulmonary abscess, various forms of cardiocirculatory disease, diaphragmatic hernia, arthritis, neuralgia, or miscellaneous other conditions. The diagnostic error is prolonged if the physician fails to become concerned by the persistence of symptoms in what he had diagnosed as a self-limited disease, or by the atypical course of some presumed chronic disease. The persistence or aggravation of the significant symptoms of cough, dyspnea, and pain should lead to a reevaluation of the clinical findings, especially the roentgenological findings. While the mistaking of a non-neoplastic lesion for a neoplasm occasionally occurs and may lead to an unnecessary operation, the opposite mistake is much more frequently made.

There is a rather generalized belief among physicians that, once a diagnosis of neoplasm has been made, immediate surgical removal is required only for those tumors believed to be

malignant. Consequently, tumors that are believed to be benign, but that are actually malignant, are often merely observed until such time as their malignant nature becomes manifest; in many cases the lesion becomes inoperable during the period of observation. There are, furthermore, real dangers in merely observing neoplasms that are actually benign when first diagnosed, as the continued enlargement of a benign tumor produces various forms of progressive disfunction of the lungs, heart, and other mediastinal organs, and as the contents of a cystic tumor, or the liquefied degenerated portion of a solid tumor, may perforate into a bronchus; the most important danger, however, is that benign neoplasms not rarely undergo malignant degeneration.

Since benign as well as malignant neoplasms should be removed promptly, the clinician's chief concern should be to determine whether a given tumor is probably completely removable and whether the patient's general condition will permit the intended operation. The operative risk should obviously be weighed against the probable malignancy or benignancy of the tumor; when the risk is sufficiently great that it would be accepted only if the tumor were malignant, a needle punch biopsy of the lesion is justifiable. A careful search should be made for evidence of metastasis or irremovable invasion of the thoracic walls. Bronchoscopy will determine whether a bronchogenic carcinoma has extended too far cephalad to be completely removed by total pneumonectomy. A tumor, especially one of the mediastinum, that might be a lymphoblastoma should be given a diagnostic test dose of deep x-ray therapy. This use of x-ray therapy is its only proper one for possibly operable intrathoracic neoplasms.

The rapid rate of growth of a tumor suggests malignancy but is not conclusive evidence since some benign tumors grow rapidly and some increase rapidly in size from hemorrhage into, or rapid increase in the liquid contents of, a cystic tumor. Furthermore, some malignant neoplasms grow very slowly and may metastasize while the original lesion is still small. Most intrapulmonary neoplasms are malignant, being carcinomas, and may be either well or poorly circumscribed, roentgenologically. Although most extrapulmonary neoplasms with well circumscribed borders are benign and those with poorly defined borders are malignant, the former are occasionally malignant and the latter benign. While the following signs are strongly suggestive of malignancy, all have occurred in cases of benign tumor: Paralysis of the phrenic, recurrent laryngeal and upper thoracic sympathetic nerves, dysphagia, dilatation of superficial veins, hemorrhagic pleural effusion, serous pleural effusion in the presence of a small neoplasm, great pain, and great loss of weight.

Since delay in removal of malignant neoplasms is not justifiable and since delay in removal of benign neoplasms exposes the patient to certain considerable risks, virtually all presumably removable intrathoracic neoplasms (with the exception of lymphoblastomas) should be operated upon soon after the diagnosis has been made, provided, of course, that the patient's general condition permits. The importance of early operation is increased by the facts that in many cases an accurate determination of malignancy or benignancy cannot be made before operation and that delay in operating in the case of those tumors that happen to be malignant often results in their becoming inoperable. Early surgery for almost all intrathoracic neoplasms will prevent many tragic deaths that would occur as a direct result of the postponement of operation in the usually unfulfilled hope that a preoperative pathological diagnosis might, somehow, soon be made. JOHN ALEXANDER.

Fig. 1.

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cta omnia adhibitis, vocatis, et rogatis

Fig. 2.

Diploma of the medical degree awarded by the University of Bologna, in 1698.

TEXTS AND DOCUMENTS

ON THE DIPLOMA AND CEREMONIES INCIDENT TO THE AWARD OF THE MEDICAL DEGREE AT BOLOGNA, ITALY, IN 1698

BLANCHE B. BOYER, and ARNO B. LUCKHARDT, M.D., Chicago, Illinois

THE modern *cap* and *gown*, official dress, at present day University convocations are, of course, curious modifications of the monastic "cowl" and "cuculla" not uncommon to this day in various materials and colors depending on the particular monastic or religious order. In the religious orders the hood or cowl is usually attached to the monk's scapular. The present day hood placed over the head and about the neck and shoulders of the present day recipient of an earned or honorary degree is a curious and misshapen homologue of the cowl attached to the scapular of which latter there is no vestigial remnant in the modern academic gown. Because of this, there was evolved in the course of time at the English speaking universities the "headgear" spoken of as the "mortarboard" or "cap" quite different from the birretum and from the earlier monk's cowl which still constitutes a serviceable protective covering for the head.

As in the case of dress, so the ceremonies incident to the investiture of a candidate on graduation have been greatly modified in the course of time, as is very evident from the diploma of the M.D. and Ph.D. degree awarded to one Franciscus Ignatius of Ravenna by the University of Bologna in 1698.

Senior author, Miss Blanche Boyer, translated from the Latin *in toto* the text of this diploma. Only such passages will be reproduced in translation as are of interest because of their novelty. The document belonging to the junior author consists of a book measuring 8½ inches by 6½ inches, bound in full calf and containing one hand colored frontispiece and four pages of text, hand printed on parchment.

Dr. Boyer, Assistant Professor of Latin, The University of Chicago (Paleographer).

In the frontispiece (Fig. 1), on the left is seated the figure of Minerva, goddess of wisdom, arts, and crafts, to whose usual attributes is added the caduceus. With the left hand she proffers the flower of learning to Bologna (the seated figure on the right, identified by the coat of arms—red cross on white background, surmounted by three gold stars—inscribed *Liberty*). Bologna with her right hand to a star in the upper center of the picture says, "If it be filled with dew" ("the flower will thrive" by context). Across the bottom of the picture runs the legend:—"Forever will live . . . . . good from me." The last page of the Latin text ends with the following transcription (Fig. 2) in translation:

"I, Antonius Felix Marsilius, Archdeacon and Senior Chancellor of the University of Bologna, testify to the acceptance of D. (ominus) Franciscus Ignatius of Ravenna in the aforesaid Faculties of Doctors. . . .

"I, Dominicus Maria, son of D. Angelus of Jordanii, Notary authorized by the Holy See, the Empire and the City of Bologna, and special secretary of His Grace and Very Reverend Lord the Archdeacon, since I was present at the foregoing request of the aforesaid eminent D. Franciscus Ignatius admitted with no dissenting vote into the aforesaid Faculties of Doctors, have therefore subscribed with my own hand to all the foregoing—drawn in the form of a public document by another's trustworthy hand—and have stamped it as required. Dated: Bologna, 24th of September, 1698."

The document commences with exuberant praise and flattery of the officials of Bologna and its uiversity, so common to that age and hardly of interest to this journal's readers.

Next the document refers in glowing terms to the doctoral candidate, "D. Mv. Franciscus Ig-

Fig. 2. Diploma of the medical degree awarded by the University of Bologna, in 1698.

Fig. 1.

natius, most eminent citizen of Ravenna" who "has undergone a difficult, rigorous and formidable examination by the Domini D. D. Doctors of said college of Liberal Arts, Sacred Philosophy and Medicine in Bologna in which said eminent D. Mr. or Dominus Franciscus Ignatius" expounded so cleverly "with unusual clarity the topics given him, by shrewd argument and clever answer after the fashion of Doctor rather than Scholar" that he was adjudged fit and satisfactory by "unanimous vote with no dissenting sign" to be "candidate for the Degree in the aforesaid Faculties."

It was also determined that his faith was beyond suspicion; for after a "prefatory discourse and information duly and rightfully received regarding the religion and Catholic faith of said eminent D. Franciscus Ignatius by the summoning and examining of reliable witnesses and by public avowal of the candidate with the oath of the Catholic faith in the usual set formula prescribed in the Bulla and Apostolic letters of Pope Pius IV of blessed memory" etc.

Everything having been found satisfactory and the candidate having requested *the insignia of the Doctorate* the procedure was as follows:

"Books of the Liberal Arts and sacred Philosophy and Medicine closed were placed in his hands, then, the same open; second, the birretum or Doctoral cap was put on his head; third, he was pledged in the name of all branches of learning with a ring of gold; then the kiss of peace and the Magisterial benediction that the same eminent D. Franciscus Ignatius, Distinguished Doctor, should be thus decorated and conspicuously rewarded in his Fatherland by Him who One and Three reigns unnumbered centuries, eternal and glorious."

Surely a most ceremonious and time consuming procedure for *one* doctor compared with the hurried and perfunctory "mass production" practice at convocations of the present time.

THE SURGEON'S LIBRARY

REVIEWS OF NEW BOOKS

IN the splendidly written book *Clinical Roentgenology of the Alimentary Tract*[1] Buckstein has drawn liberally not only from the literature but particularly from his rich experience in the field of gastroenterology. In the preface and throughout the text the importance of the correlation of the clinical, etiological, and pathological with the roentgenological aspects of diseases of the alimentary tract is stressed. Since roentgenography has become such an essential procedure in the diagnosis of lesions of the alimentary tract, it is important that any physician having to deal with the diagnosis and treatment of such lesions have a knowledge of the potentiality of this method. In many instances the diagnosis will be evident on the roentgenogram but not infrequently in the borderline cases only a correlation of the clinical and the roentgen findings will lead to a correct interpretation.

This volume includes not only an excellent presentation of the roentgenology of the digestive tube, the biliary tract, the spleen, liver, and pancreas, but also pertinent details as to roentgenographical technique and procedures so important in an adequate examination. The importance of using both fluoroscopy and radiographical methods are discussed and emphasized. There should be no controversy as to which method is most important as both are essential for an adequate and complete examination of the alimentary tract. Throughout the volume the author has presented numerous interesting case reports which help to emphasize the roentgen findings. The illustrations are numerous, well chosen, and beautifully reproduced, and add greatly to the clarity of the discussion.

This comprehensive, easily readable, and dependable volume should prove most helpful and instructive, not only to the roentgenologist and gastroenterologist but to the physician in general practice whose needs the author has kept in mind throughout the presentation. It is recommended without reservation and merits wide distribution.

EARL E. BARTH.

THE volume by Dr. Salter[2] contains an authoritative presentation of present day knowledge of iodine in physiology and biochemistry. The text includes the author's very extensive work in this field so that it is a most valuable monograph. In addition, it gives a review of physiological and chemical literature on iodine. More than 600 references are listed. Of necessity, thyroid gland physiology is thoroughly discussed, so that, as the preface indicates, "an important bridge between biochemistry and endocrinology" is provided. Recent studies of radioactive iodine as a means of elucidating iodine metabolism are discussed. Blood iodine and iodine balance studies in various thyroid diseases are presented and much of the modern data are reproduced in tabular and graphic form. Iodine metabolism in other diseases and physiological states is also reviewed. The technique of iodine determination in biological material is given in detail. Hence, this volume is essential to students of clinical endocrinology as well as to biochemists. It cannot be recommended too highly.

PAUL STARR.

THE eighth edition of *Williams Obstetrics*[3] has recently been made by Stander, this being his second revision of the well known text. The book has been almost completely rewritten and little of the original text remains unaltered. Three new chapters have been added: "Diseases and Abnormalities of the Newly Born Child," "Classification of Abnormal and Contracted Pelves," and "Sudden Death and Maternal Mortality." This text has been accepted as a classic for so long that an extended review is unnecessary. The field of obstetrics has been completely covered, and all recent advancements made in the field are incorporated in the text. Controversial theories and different teachings are presented, and the author's own views and teachings are definitely stated.

In the chapter on classification of abnormal and contracted pelves, Stander presents the modified Williams classification, which is based on an etiological basis. The morphological classification of Caldwell and Moloy based on sexual and anthropological characteristics is presented, as is also the Thoms classification of pelves. A new classification of contracted and abnormal pelves is proposed by Stander; this classification is based on a combined morphological and etiological classification and is arranged on a basis of inherited, sexual, developmental, growth, disease, and traumatic factors. Roentgenological pelvimetry is discussed in detail and techniques of the procedure are also described in full detail.

In the new chapter, diseases and abnormalities of the newborn child, abnormalities, injuries, and diseases occurring during pregnancy, labor, and shortly thereafter are discussed.

[1] CLINICAL ROENTGENOLOGY OF THE ALIMENTARY TRACT. By Jacob Buckstein, M.D., Philadelphia and London: W. B. Saunders Co., 1940.

[2] THE ENDOCRINE FUNCTION OF IODINE. By William Thomas Salter. Cambridge, Massachusetts: Harvard University Press, 1940.

[3] WILLIAMS OBSTETRICS. By Henricus J. Stander, M.D., F.A.C.S. 8th ed. New York and London: D. Appleton-Century Co., 1941.

The chapter on the toxemias of pregnancy has been revised, and the toxemias are classified and discussed according to the new classification of the American Committee on Maternal Health. Hypertensive disease and renal disease complicating pregnancy, while not true toxemias of pregnancy, are discussed in this chapter because of their similarity in several respects to the toxemias. Clinical and laboratory methods of diagnosis and differentiation of this important group are given in detail, and their treatment, in view of our present knowledge of these conditions, is thoroughly presented.

The chapters on operative obstetrics are most complete and operative procedures are well visualized by means of photographs and drawings. The chapter on amnesia, analgesia, and anesthesia in labor gives the history and progress of the preparations used from their inception to the present. Their methods of use, advantages, and disadvantages are fully stated and details of application employed at present in the New York Lying-In Hospital are outlined.

This is a most valuable book for students and practitioners, and essential to the library of anyone teaching obstetrics. THEODORE J. MORRIS.

WELL known for his contributions to the knowledge of blood coagulation, Dr. Nygard has recently written *Hemorrhage Diseases*.[1] Much of the present work centers around the determination of blood coagulability by a photo-electric method developed by the author. The principle of the method is the measure of light transmission through recalcified blood plasma during fibrin formation. Automatic graphic recordings are made of the progressive reaction. From the curves, it is possible to determine the time of the first formation of fibrin, the duration and velocity of fibrin formation and the appearance of retraction of the clot. This method obviously gives much more accurate and detailed information than clinical methods for blood coagulation now in use.

In the first part of the book, the writer outlines the methods of determining coagulability of the blood and describes in detail the principles of the photo-electric method. In part II are included experimental investigations on the coagulability of blood plasma, the interaction of thrombin and fibrinogen and the coagulant effect of thromboplastin. The photo-electric method helps greatly to clarify the mechanism of blood coagulation. In the last part of the book there is a complete classification of "hemorrhagic manifestations." The comprehensive chapters on hemophilia, thrombocytopenic purpura, vitamin K, jaundice, and hemorrhagic disease of the newborn include many interesting and original observations on the coagulation defect in these conditions.

More complicated sections of the book are simplified by many well chosen illustrations and tables. Complete references are given at the end of each chapter. This excellent volume should be of great value to both physiologists and clinicians interested in the problem of blood coagulation.

HOWARD L. ALT.

ACCORDING to its preface, *Spermatozoa and Sterility* by Abner I. Weisman[2] is an effort to bring to the attention of those interested in sterility the tremendous importance of semen, spermatozoa, and spermigation in the female by concentrating in one volume all of the available information on the subject up to the present time. In this effort, Dr. Weisman has very largely succeeded.

Seventeen of the book's 27 chapters have to do with the seminal fluid or its constituents. The chemistry, metabolism, and reaction of spermatozoa to external stimuli are adequately discussed. Methods for obtaining and evaluating spermatozoa are given in excellent detail. Specific instructions for collecting and counting spermatozoa, and for studying spermatozoal morphology are presented in a simple, lucid manner. In a discussion of the interpretation and evaluation of seminal analysis the author correctly directs attention to the fact that a single seminal specimen does not, in itself, give a complete picture of the human male's procreative ability. Thorough physical examination and repeated study of the seminal fluid are essential. While the value of many of the procedures included as auxiliary tests of spermatozoal function would be questioned by some gynecologists, yet they are rightfully included in a comprehensive compilation of the literature.

The problem of artificial impregnation with either the husband's or a donor's semen is presented not only from the medical but also from the legal aspect. The pitfalls inherent in such unnatural procedures are pointed out and measures directed toward decreasing some of the dangers associated with artificial insemination are given. Special chapters are devoted to pregnancy tests, spermatoxins, control of sex, and theoretical factors in fertility and sterility. Only two rather brief chapters deal in a general way with the causes of sterility in the male and with the treatment of impaired spermatogenesis.

The text is an excellent source book not only for the gynecologist and the urologist but also for the experimental laboratory worker as well. It is somewhat voluminous to be considered an ideal manual for the general practitioner. The text contains references to and excerpts from most of the authoritative investigations of human sterility. The monumental work of Retzius has been emphasized but it is to be regretted that Gatenby's and Beams' newer observations on spermatozoal morphology were overlooked. Throughout the text the author has been meticulous in giving credit for original work. His book is a timely reminder of the need for a careful study of the husband's seminal fluid in human sterility problems. JOHN W. HUFFMAN.

[1] HEMORRHAGIC DISEASES; PHOTO-ELECTRIC STUDY OF BLOOD COAGULABILITY. By Kaare K. Nygaard, M.D. St. Louis, Mo.: The C. V. Mosby Co. 1941.

[2] SPERMATOZOA AND STERILITY; A CLINICAL MANUAL. By Abner I. Weisman, M.D. New York and London: Paul B. Hoeber Inc., 1941.

CLINICAL CONGRESS OF AMERICAN COLLEGE OF SURGEONS

EVARTS A. GRAHAM, St. Louis, *President*
W. EDWARD GALLIE, Toronto, *President-Elect*

Committee on Arrangements
LELAND S. MCKITTRICK, *Chairman;* RICHARD H. SWEET, *Secretary*

PROGRAM FOR THE 1941 CLINICAL CONGRESS IN BOSTON

THE fellows of Boston and vicinity will sponsor the thirty-first annual Clinical Congress of the American College of Surgeons November 3 to 7, when the facilities of that great medical center will be employed to provide a clinical program of exceptional interest. The co-operation of Boston's three medical schools and some 30 approved hospitals is assured. Operative clinics and demonstrations in the hospitals will cover many phases of general surgery and the surgical specialties. Leaders of the profession from many sections of the country will participate in the panel discussions and other scientific meetings at the headquarters hotels. The Hospital Standardization Conference will attract visitors of outstanding reputation in the hospital field. The program will be one of broad scope and interest, and it may be confidently expected that the notable contributions to medicine and surgery made at previous meetings in Boston, beginning in 1915, will be repeated.

CLINICAL PROGRAM

The clinical program for the five-day meeting is being arranged under the leadership of a strong and representative Committee on Arrangements. An extensive schedule of clinics, symposia, and demonstrations in the hospitals and medical schools will, as usual, be the major feature of the Congress. Visiting surgeons will be given opportunity to observe and obtain first-hand information on a wide variety of surgical and related subjects, in the favorable environment of excellent hospital facilities. In the operative clinics, surgical technique, use of operating room equipment, and organization of personnel will be demonstrated. The symposia and non-operative clinics will deal with the broader but equally important aspects of diagnosis, related medical treatment, pre-operative preparation and post-operative care. The discussions will be augmented by the presentation of clinical material. Diagnostic methods will be demonstrated by various hospital departments. Specialists in many other fields will participate in the clinical program at the hospitals in order that the subjects may receive consideration from different viewpoints. The preliminary clinical program appearing in the following pages is to be revised and amplified in the weeks preceding the Congress.

The medical schools and their affiliated hospitals have developed a series of exhibits presenting their work in clinical investigation and the latest advances in surgical research. These exhibits will be available for study in the local hospitals during the entire meeting. Basic science departments of the medical schools will contribute to the program by arranging exhibits and demonstrations related to surgical practice. Thus the Boston institutions have much to offer in the way of a highly concentrated postgraduate course in surgery and allied subjects.

The clinical program is so arranged that the visiting surgeon may readily select the clinics which he wishes to attend. The schedule for each hospital will cover subjects in general surgery, obstetrics and gynecology, fractures, orthopedic surgery, thoracic surgery, neurosurgery, genito-urinary surgery, ophthalmology, otorhinolaryngology. Presentation of subjects under these classifications is so correlated that he will have the opportunity to devote his time continuously to clinics dealing with the specialty in which he is most interested. The clinical program as published daily will be arranged according to these classifications. Each afternoon during the Congress, the

CLINICAL CONGRESS PROGRAM IN BRIEF

Monday, November 3

9:30 Film Exhibition, General Surgery, Statler.
10:00 Film Exhibition, Eye, Ear, Nose and Throat Surgery, Statler.
10:00 Hospital Conference, Copley-Plaza.
1:30 Panel Discussions (4), Statler.
2:00 Film Exhibition, General Surgery, Statler.
2:00 Clinics in Boston Hospitals.
2:00 Hospital Conference, Copley-Plaza.
3:00 Assembly of Initiates, Reception, Hancock Hall.
3:30 Panel Discussions (4), Statler.
8:00 Presidential Meeting and Convocation, Symphony Hall.

Tuesday, November 4

7:45 Breakfast Conference, Copley-Plaza.
9:00 Clinics in Boston Hospitals.
9:00 Film Exhibition, Eye, Ear, Nose and Throat Surgery, Statler.
9:30 Hospital Conferences (2), Copley-Plaza.
9:30 Forum on Fundamental Surgical Problems, Copley-Plaza.
11:00 Hospital Conferences (2), Copley-Plaza.
11:00 Group Conferences, Eye, Ear, Nose and Throat Surgery (2), Statler.
12:00 Film Exhibition, General Surgery, Statler.
1:30 Panel Discussions (3), Statler.
2:00 Clinics in Boston Hospitals.
2:00 Hospital Conference, Copley-Plaza.
2:00 Cancer Symposium, Copley-Plaza.
3:30 Panel Discussions (4), Statler.
5:15 Clinical Pathological Conference, Statler.
6:00 Film Exhibition, General Surgery, Copley-Plaza.
7:00 Film Exhibition, Eye, Ear, Nose and Throat Surgery, Statler.
8:00 Hospital Conference, Copley-Plaza.
8:00 Scientific Session, General Surgery, Copley-Plaza.
8:00 Scientific Session, Ophthalmology, Statler.
8:00 Scientific Session, Otorhinolaryngology, Statler.

Wednesday, November 5

7:45 Breakfast Conference, Copley-Plaza.
9:00 Clinics in Boston Hospitals.
9:00 Film Exhibition, Eye, Ear, Nose and Throat Surgery, Statler.
9:30 Hospital Conference, Copley-Plaza.
9:30 Forum on Fundamental Surgical Problems, Copley-Plaza.
9:30 State and Provincial Judiciary Committees, Hancock Hall.
10:00 State and Provincial Executive Committees, Hancock Hall.
10:30 State and Provincial Credentials Committees and Committees on Applicants, Hancock Hall.
11:00 Group Conferences, Eye, Ear, Nose and Throat Surgery (2), Statler.
12:00 Film Exhibition, General Surgery, Statler.
12:00 Meeting of Board of Governors, Copley-Plaza.
1:30 Panel Discussions (4), Statler.
2:00 Clinics in Boston Hospitals.
2:00 Symposium on Fractures and Other Traumas, Copley-Plaza.
2:30 Hospital Conference, Demonstrations in local hospitals.
2:30 Film Exhibition, General Surgery, Statler.
3:30 Panel Discussions (4), Statler.
5:15 Clinical Pathological Conference, Statler.
6:00 Film Exhibition, General Surgery, Copley-Plaza.
7:00 Film Exhibition, Eye, Ear, Nose and Throat Surgery, Statler.
8:00 Scientific Session, General Surgery, Copley-Plaza.
8:00 Joint Session, Sections on Ophthalmology and Otorhinolaryngology, Statler.
8:00 Hospital Conference, Motion Pictures, Copley-Plaza.

Thursday, November 6

7:45 Breakfast Conference, Copley-Plaza.
9:00 Clinics in Boston Hospitals.
9:00 Film Exhibition, Eye, Ear, Nose and Throat Surgery, Statler.
9:30 Forum on Fundamental Surgical Problems, Copley-Plaza.
9:30 Hospital Conference, Copley-Plaza.
11:00 Hospital Conference, Copley-Plaza.
11:00 Group Conferences, Eye, Ear, Nose and Throat Surgery (2), Statler.
12:00 Film Exhibition, General Surgery, Statler.
1:30 Annual Meeting of Governors and Fellows, Copley-Plaza.
2:00 Clinics in Boston Hospitals.
2:00 Hospital Conference, Copley-Plaza.
3:30 Panel Discussions (5), Statler.
3:30 National and Regional Fracture Committees, Copley-Plaza.
5:15 Clinical Pathological Conference, Statler.
6:00 Film Exhibition, General Surgery, Copley-Plaza.
7:00 Film Exhibition, Eye, Ear, Nose and Throat, Statler.
8:00 Scientific Session, General Surgery, Copley-Plaza.
8:00 Scientific Session, Ophthalmology, Statler.
8:00 Scientific Session, Otorhinolaryngology, Statler.

Friday, November 7

9:00 Clinics in Boston Hospitals.
9:30 Film Exhibition, Eye, Ear, Nose and Throat Surgery, Statler.
9:30 Film Exhibition, General Surgery, Statler.
9:30 Forum on Fundamental Surgical Problems, Copley-Plaza.
1:30 Panel Discussions (4), Statler.
2:00 Clinics in Boston Hospitals.
2:00 Film Exhibition, General Surgery, Statler.
3:30 Panel Discussions (3), Statler.
8:00 Boston Surgical Society, John Hancock Hall.

complete detailed clinical program for the succeeding day will be posted in the form of bulletins at headquarters in the Statler Hotel. On the following morning this final program will be distributed in printed form. As much information as it is possible to obtain in advance is being published in the preliminary programs and the general program, but it is necessary that those who attend the Congress make their clinic ticket selections from the daily bulletins. This will facilitate proper selections and distribution of clinic tickets.

PRESIDENTIAL MEETING AND CONVOCATION

On Monday evening in Symphony Hall, the combined Presidential Meeting and Convocation will be opened by an impressive processional of

the officers, regents, and honorary guests. Welcome will be extended to the assembly by the chairman of the local Committee on Arrangements, Dr. Leland S. McKittrick. Honorary guests will then be introduced, following which Dr. Evarts A. Graham, the retiring president, will deliver the presidential address. Other events on the program of this meeting will be the inaugural ceremony for the incoming officers, the presentation of the initiates for fellowship, and the awarding of the Medical Records Prize.

SCIENTIFIC SESSIONS

The scientific meetings, to be held on Tuesday, Wednesday, and Thursday evenings in the ballroom of the Copley-Plaza Hotel, will be addressed by surgeons and specialists recognized as authorities in their respective fields. Complete programs for all scientific sessions will be found in the following pages. The annual oration on surgery will be given by Dr. W. Edward Gallie, Toronto, Ontario, on Tuesday evening.

The Board of Regents of the College has given careful attention to the selection of speakers and subjects for these evening sessions so that a well-rounded program introducing the newer developments in general surgery and the surgical specialties may be assured.

Great interest has been demonstrated during recent years, at both the Clinical Congress and sectional meetings of the College, in the panel discussions held at headquarters each afternoon. For the Boston meeting a schedule of thirty-five of these sessions has been planned for Monday, Tuesday, Wednesday, Thursday, and Friday. Recognized authorities in the fields under discussion will lead these panels, and with the aid of collaborators especially qualified will present the fundamental facts and direct the discussions in such a way that different viewpoints may be expressed. These sessions afford an opportunity for a larger number of surgeons to participate in the discussions and learn of the ideas and experiences of others. A ten-minute presentation of the subject is made by the leader, followed by brief remarks by the collaborators on various phases of the topic. As the major feature of each session, general discussion from the floor will be encouraged. This type of program will permit more informal discussion than usually takes place in larger general meetings. The subjects of the panels are carefully selected in order to cover many pertinent problems in various fields of surgery. It is suggested that surgeons who have questions which they would like to have considered in any panel discussions they plan to attend, should communicate in advance with the panel leaders so that if possible such questions may be specially incorporated in the program. This will help to assure discussion of points which those in attendance would like to have clarified.

Important features of the afternoon meetings at the headquarters hotels are a symposium on "Cancer" on Tuesday and one on "Fractures and Other Traumas" on Wednesday. It should be noted also that special panel discussions deal with "The Organization and Conduct of Cancer Clinics in Hospitals," and "Graduate Training in Surgery." Because of the wide interest in these subjects, it is expected that these meetings will attract a large attendance.

In formulating the program it is the aim of the Board of Regents and the committee to make it possible for each surgeon to learn of the newer developments in his field of practice. The subjects presented in the hospital clinics, panel discussions, and other scientific meetings will appeal to the surgical specialist as well as to the general surgeon. Each feature of the meeting has been carefully planned with this idea in mind. The detailed programs for these sessions will be found on the following pages.

FORUM ON FUNDAMENTAL SURGICAL PROBLEMS

A new feature to be introduced at the Clinical Congress in Boston is a "Forum on Fundamental Surgical Problems," the plans for which have been formulated by a committee under the chairmanship of Dr. Owen H. Wangensteen, of Minneapolis. In authorizing this new program it is the purpose of the Board of Regents to enable younger men representing various university departments of surgery to present the important results of their clinical and experimental research work before one of the larger surgical meetings. Heretofore, there has been limited opportunity for the younger men, many of whom have not yet qualified for membership in the principal surgical societies, to present their original work and ideas before representative surgical bodies. As surgery is concerned today not only with anatomy and pathology but to a growing extent with physiology, chemistry and physics, it is felt that the Clinical Congress program will be further broadened by the presentation of the best of this material and that surgeons attending the Congress may distinctly benefit therefrom.

Sessions will be held on Tuesday, Wednesday, Thursday and Friday mornings in the ballroom of the Copley-Plaza Hotel with presentations of original and experimental work related to general surgery and the surgical specialties. There will be

no prepared discussions but opportunity will be afforded those who attend to ask questions. Presentations will be limited to ten minutes each. It is expected that some of the best examples of new and highly constructive developments in surgery will be presented, and there is every indication that a live and active surgical forum in which younger men may participate will constitute one of the most important activities of the Congress in the future.

The committee in charge has been highly pleased with the response to this idea. Over 125 abstracts of subjects available for presentation have been submitted, and from this material the committee will select the most outstanding for the forum. The subjects, together with brief abstracts, will be published in the final program of the Clinical Congress.

OPHTHALMOLOGY AND OTORHINOLARYNGOLOGY

The Congress will include an extensive program of scientific sessions and clinical demonstrations for ophthalmologists and otorhinolaryngologists. Separate sessions scheduled for Tuesday and Thursday evenings will include papers and symposia on pertinent subjects in these fields. The leaders will direct the discussions in the meetings so as to cover many phases of a general subject in each of the specialties. Those attending will have an opportunity to express their viewpoints. On Wednesday evening, there will be a joint meeting of ophthalmologists and otorhinolaryngologists with a symposium on "Neurosurgery as Related to the Eye and Ear." This type of program promises to attract wider interest than the presentation of formal papers.

Clinics in the Boston hospitals each morning and afternoon for the visiting ophthalmologists and otorhinolaryngologists will demonstrate surgical work of great variety. On Tuesday, Wednesday, and Thursday mornings, in addition to the hospital program, there will be clinical conferences for each group of specialists at the Statler Hotel. These are being arranged so that the leader will briefly survey the field for discussion in a ten-minute period. The larger meeting will then be broken up into small groups, limited to 20, and under separate leaders where there will be an opportunity for everyone to ask questions and participate in the discussion. Each visitor will select in advance the smaller group which he wishes to attend. In this manner, the general subject may be reviewed thoroughly and each visitor will benefit by the experiences and ideas of his colleagues. The morning and evening meetings will be preceded by the showing of selected motion picture films on subjects related to ophthalmology and otorhinolaryngology. The detailed programs of these special meetings appear in the following pages.

SPECIAL MEETING OF BOSTON SURGICAL SOCIETY

On the evening of Friday, November 7, in John Hancock Hall, which is located midway between the Statler and Copley-Plaza Hotels, the Boston Surgical Society will hold a special meeting at which all fellows of the College and other surgeons attending the Clinical Congress are invited to be present. The occasion of this special meeting of the society is the presentation of the Bigelow Medal to Dr. Allen O. Whipple, of New York, for his outstanding contributions to the advancement of surgery. The program follows:

Introduction: Irving J. Walker, M.D., President.

Address: "Present-day Surgery of the Pancreas," Allen O. Whipple, M.D.

Presentation of the Bigelow Medal: David Cheever, M.D.

CLINICAL PATHOLOGICAL CONFERENCES

Selected fresh surgical specimens from the operative clinics of the various hospitals will be demonstrated each day with the aid of the Zeiss epidiascope in the Georgian Room of the Statler Hotel from 5:15 to 6:30 p.m. This is possible through the co-operation of the pathologists of the hospitals taking part in the Congress. The method of projection will enable all to see clearly the pathological processes involved.

By the use of fresh, rather than preserved material, chosen from each day's operative clinics, the surgeons interested in the various types of operation done during the day will be able to see the underlying pathology demonstrated and to hear a discussion of the clinical implications. Each conference will be conducted by a different pathologist from one of the co-operating hospitals or medical schools.

PUBLICATION OF PROCEEDINGS

As in former years, the formal papers which are presented at the scientific sessions of the Congress will be presented in a special issue of the official journal of the College, "SURGERY, GYNECOLOGY, AND OBSTETRICS," published in February following the meeting. This issue is furnished without additional charge to all fellows, junior candidates, and others who register for the Congress as invited guests. The papers which are presented in connection with the Hospital Standardization Conference are published in subsequent issues of the *Bulletin* of the American College of Surgeons.

ASSEMBLY OF INITIATES

The Assembly of Initiates will be held on Monday afternoon in John Hancock Hall. Dr. Evarts A. Graham, president, and Dr. Albert O. Singleton, Galveston, Texas, vice-president of the College, will preside. Dr. Bowman C. Crowell and Dr. Malcolm T. MacEachern will discuss "The Program of the American College of Surgeons." Initiates will then recite the fellowship pledge and greetings will be extended by Dr. W. Edward Gallie, Toronto, president-elect of the College. Closing remarks will be made by Dr. Irvin Abell, Chairman of the Board of Regents.

NATIONAL AND REGIONAL FRACTURE COMMITTEES

The meeting of the National and Regional Fracture Committees, with Dr. Robert H. Kennedy, of New York, chairman of the National Committee, presiding, will be held on Thursday afternoon at the Copley-Plaza Hotel, when this assembly of surgeons will discuss the activities of the respective groups. Working in co-operation with the American Red Cross, other local organizations and public officials, these committees have exerted great influence in improving methods and facilities for the transportation of the injured. They have also made a concerted effort to improve the treatment of fractures in the hospitals of many communities. Pertinent problems will be discussed at the meeting of these committees.

ANNUAL MEETING OF COMMITTEES

The annual meetings of the State and Provincial Judiciary, Credentials, and Executive Committees will be held on Wednesday morning in John Hancock Hall. These committees have an important function to perform in the College. The Credentials Committees and the Committees on Applicants constitute one of the largest and most carefully deliberate accrediting bodies which exist in the medical profession. Through this organization the standards of fellowship are maintained and each fellow of the College has a definite responsibility in this work. All members of these committees are urged to attend this important meeting.

ANNUAL MEETING OF FELLOWS

The annual meetings of the governors and fellows will be held on Thursday afternoon in the ballroom of the Copley-Plaza Hotel. The American College of Surgeons has been a potent force which has not only raised the professional and ethical standards of surgery, but has also promoted good hospitalization and general improvement in the practice of medicine in the United States and Canada. Each fellow of the College has a personal part in this work and may extend its influence materially in his local community. Hospital standardization alone offers him unlimited opportunity to provide better medical care for his patients in the hospital in which he works through continuous progress in applying the principles of the minimum standard. Other activities of the College have also received wide recognition by professional groups and the public as well, and together they present a vast educational program.

The annual meeting of the fellows affords officials of the College an opportunity to report on the work of the organization and to receive suggestions from those who have made possible the conduct of these activities. Every fellow of the College will want to attend this important meeting.

MEDICAL MOTION PICTURES

Visual education plays an increasingly important rôle in the education of the surgeon. In recognition of this fact, an enlarged program of surgical motion pictures will be presented at headquarters which will include the latest available films on a variety of subjects. Schedules will be arranged so as not to conflict with the clinical program at hospitals or the scientific sessions, and will include both sound and silent, and color films, all of which have been approved by the Committee on Medical Motion Pictures of the College.

HOSPITAL CONFERENCES

The Congress will open on Monday morning at 10:00 o'clock with the first session of the twenty-fourth annual Hospital Standardization Conference in the ballroom of the Copley-Plaza Hotel. The report of the 1941 hospital standardization survey—official announcement of the list of approved hospitals and hospitals approved for graduate training in surgery—will be made at this session. Dr. Evarts A. Graham, of St. Louis, president of the College, will preside. In addition there will be interesting discussions on the subjects of "Medical Preparedness," "The Preservation of Our Voluntary System of Hospitals," and "Maintaining the Control and Quality of the Professional Work in the Hospital." On Monday afternoon a panel discussion will deal with the timely subject of "Meeting the Problems of Rendering Adequate Care of the Patient and Maintaining Quality Standards of Service During the Present Period of Preparedness and National Emergency," with Dr. Nathaniel W. Faxon, of Boston, presiding.

Of special interest to surgeons, as well as hospi-

tal personnel, will be a series of panel discussions dealing with the efficient organization of the hospital. On Tuesday morning four panel discussions will deal with the essentials for efficiency of the hospital in (1) the Obstetrical Department, Dr. Harvey R. Matthews, Brooklyn, presiding; (2) the Orthopedic Department, Dr. Henry H. Kessler, Newark, New Jersey, presiding; (3) Organization and Functioning of the Medical Staff, Dr. Joseph C. Doane, Philadelphia, presiding; and (4) the Department of Anesthesia, Dr. Ralph T. Knight, Minneapolis, presiding. Two additional panel discussions will be held on Thursday morning; one on the "Essentials Pertaining to an Efficient Department of Urology" will be led by Dr. George F. Cahill, New York, and the other, "Essentials Pertaining to an Efficient Department of Ophthalmology and Otolaryngology," by Drs. Conrad Behrens and Marvin Jones, of New York.

In preparing this portion of the program, organizations representing the specialty groups have been invited to participate. Special consideration will be given in this series of meetings to the important subject of graduate training in surgery. It is hoped that through this means the fellows of the College may become better acquainted with its program of hospital activities, which is aimed at improvement of the environment in which the surgeon is working—the hospital.

On Thursday afternoon, Dr. Robin C. Buerki, of Philadelphia, will lead a round-table conference on "Pertinent Problems Emanating from a Complete Survey of the Hospital Field." An ample opportunity will be given those present to submit questions which are not on the special program.

The entire program as detailed in the following pages will hold great interest for members of medical staffs, trustees, administrators, and other hospital personnel. On Tuesday afternoon several pertinent subjects will be presented including "The Organization of a Blood Transfusion Department in a General Hospital," by Drs. Leo M. Zimmerman and Sidney O. Levinson, Chicago. Following these papers a sound motion picture on the subject of "The Proper Use and Care of Hypodermic Needles" will be shown. Tuesday evening's session will be of particular interest to hospital trustees when the subject of the panel discussion will be "The Hospital Trustee in His Relationships and Responsibilities," led by Raymond P. Sloan, of New York.

On Wednesday morning, a joint session with the American Association of Medical Record Librarians will be held, with Dr. Robin C. Buerki, of Philadelphia, presiding. Wednesday evening will be given over to the showing of medical motion pictures for hospital personnel, including a new film, "White Battalions—Serving All Mankind."

For Wednesday afternoon a series of demonstrations in the local hospitals has been arranged. Those who are acquainted with Boston are fully aware of the inspiration furnished by the privilege of inspecting one of the country's greatest hospital centers. Those who have not had that privilege have a memorable experience in store.

At both the Clinical Congress and the sectional meetings of the College during recent years, the breakfast hospital conferences have been especially well attended. In Boston, such a conference will be held on Tuesday morning when there will be a "Forum on Fundamental Problems of Hospital Administration," sponsored by the American College of Hospital Administrators, in co-operation with the American College of Surgeons.

On Wednesday morning, the breakfast conference will be sponsored by the Boston Medical Record Librarians Association, in co-operation with the College, when there will be held a "Forum on Fundamental Considerations in the Art and Science of Medical Record Keeping." The Thursday morning breakfast conference will be devoted to hospital public relations. The plan is to allow each speaker ten minutes to present his subject, following which everyone will be called upon to give his reaction to the problem under discussion. Attendance at these meetings will necessarily be limited and arrangements will be made for advance registration.

One of the most important features of the hospital conference will be the daily consultation service from 4:30 to 6:00 p.m. This service affords an opportunity for everyone attending the meeting to consult with recognized leaders in the hospital field on any question or problem on which specific information is desired.

ADVANCE REGISTRATION

The hospitals and medical schools of the Boston area afford accommodations for a large number of visiting surgeons, but to insure against overcrowding, attendance at the Congress will be limited to the number that can be comfortably accommodated at the clinics. The limit of attendance will be based on a survey determining the available facilities in the participating hospitals and schools. It is expected, therefore, that surgeons who wish to attend the Congress will register in advance. A registration fee will be required in order to provide funds with which to meet expenses of the meeting. A formal receipt

will be issued to each surgeon registering in advance which will be exchanged for a general admission card upon presentation at headquarters during the Congress. This card, which is not transferable, must accompany all requests for clinic tickets and be presented for admission to the scientific sessions.

A resolution adopted by the Board of Regents provides that the registration fee for fellows of the College and endorsed junior candidates shall be $5.00; that no fee for the 1941 Clinical Congress shall be required of initiates (class of 1941); that the fee for surgeons who are not fellows, attending as invited guests of the College, shall be $10.00.

As in previous years, admission to clinics and demonstrations in the hospitals and certain of the scientific meetings at headquarters will be controlled by means of tickets. This plan provides for the distribution of visiting surgeons at the various clinics and other meetings, and helps to insure against overcrowding. The number of clinic tickets issued will be limited in each case to the capacity of the room in which the clinic is held. It should be pointed out that the clinical program as published in the following pages and also in the official program to be distributed at the Congress, obviously cannot include all of the detailed information regarding operative clinics and demonstrations scheduled for the various hospitals. The complete and final program will be provided from day to day, posted in the form of bulletins at headquarters each afternoon for the succeeding day, and published in the *Daily Clinical Bulletin* for distribution each morning. Visiting surgeons are urged to consult the bulletins posted at headquarters and the *Daily Clinical Bulletin* in selecting the clinics they wish to attend, and in making requisitions for clinic tickets. Co-operation in making the clinic ticket plan a success will be greatly appreciated.

HEADQUARTERS AND TECHNICAL EXHIBITION

Headquarters for the Congress will be established at the Statler and Copley-Plaza Hotels where there are ample facilities for accommodating the Congress. All of the public rooms have been reserved for the exclusive use of the College during the five-day meeting. Registration for visiting surgeons and their ladies, the Technical Exhibition, executive offices of the College, and certain scientific sessions and medical motion picture exhibitions will be located in the Statler Hotel. Registration for those attending the hospital conference, exhibits of College activities, and the morning, afternoon, and evening hospital conferences will be located in the Copley-Plaza Hotel. The evening scientific sessions on Tuesday, Wednesday, and Thursday will also be held at the Copley-Plaza.

The Technical Exhibition, registration desk for attending surgeons, clinic ticket and information bureau will be located in the ballroom on the mezzanine floor of the Statler Hotel. Additional space for the exhibition will be provided in the Assembly Room and the mezzanine lounge adjacent to the ballroom. Leading manufacturers of surgical instruments and supplies, sutures, dressings, pharmaceuticals, operating room equipment of all kinds, and publishers of medical books will be represented in the exhibition. It will provide an opportunity for surgeons and hospital visitors to carefully inspect the finest modern products of all those industries which are helping to improve the service of hospitals and surgeons.

REGISTRATION FOR VISITING LADIES

A registration desk and information bureau for the visiting ladies will be established on the mezzanine floor of the Statler Hotel, under the sponsorship of the ladies of the Women's Auxiliary. An invitation is extended to wives, daughters, and friends of fellows attending the Clinical Congress to register and avail themselves of the information bureau which will be open daily during the Congress, offering assistance in locating visiting friends, shopping, sight-seeing and other matters.

SPECIAL TRAIN TO BOSTON VIA NEW YORK CENTRAL LINES

For the convenience of surgeons living in the central, western, southwestern and northwestern states, who will attend the Clinical Congress in Boston, arrangements are being made by the New York Central Lines to provide a special train leaving Chicago at 1 p.m. on Sunday, November 2, arriving in Boston at 9 a.m. on Monday. This special train will be equipped with cars of latest design, including club, lounge, observation, room cars of all types and standard Pullman cars. No extra fare will be charged. Fellows are urged to make their reservations for this special train at the earliest possible date, making application to the office of the Assistant General Passenger Agent, New York Central Lines, LaSalle Street Station, Chicago.

RAILROAD FARES

As no special rates have been authorized by the railroads of the United States or Canada for the 1941 Clinical Congress in Boston, in accordance with the policy adopted by the railroads, no certificates will be required. However, round-trip

tickets to Boston, sold at less than double the regular one-way fare, will be available from points in the United States and Canada. Return limit privileges of such round-trip tickets are liberal but are not uniform in all sections of the country. In most instances the return limit is 30 days or more. Surgeons planning to attend the Congress should consult local ticket agents several days in advance of the date of the meeting for complete information as to fares, routes, stopover privileges, etc.

BOSTON HOTELS AND THEIR RATES

In addition to the headquarters hotels, the Statler and Copley-Plaza, there are a number of first-class hotels within a short distance of headquarters. These will provide ample facilities at reasonable rates. It is suggested that reservations for hotel accommodations be made well in advance of the meeting. The following hotels are recommended by the committee:

	Minimum Rate with Bath	
	Single	Double
Bellevue, 21 Beacon Street	$3.00	$4.50
Bradford, 275 Tremont Street	3.00	4.50
Brunswick, 520 Boylston Street	3.00	4.00
Buckminster, 645 Beacon Street	2.50	4.00
Copley-Plaza, 138 St. James Avenue	4.00	6.00
Copley Square, 47 Huntington Avenue	2.75	4.00
Kenmore, 490 Commonwealth Avenue	3.50	5.00
Lenox, Exeter Street	3.00	3.50
Lincolnshire, 20 Charles Street	3.00	5.00
Parker House, 60 School Street	3.50	5.00
Ritz-Carlton, 15 Arlington Street	5.00	8.00
Sheraton, 91 Bay State Road	3.00	5.00
Somerset, 400 Commonwealth Avenue	3.50	6.00
Statler, Park Square	3.50	5.00
Touraine, 62 Boylston Street	3.50	5.00
Vendome, 160 Commonwealth Avenue	3.00	4.00
Westminster, 124 St. James Avenue	2.50	4.00

COMMITTEE ON ARRANGEMENTS

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Frederick H. Verhoeff
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Shields Warren

HOSPITALS AND REPRESENTATIVES

Beth Israel Hospital—Jacob Fine
Beverly Hospital—Richard E. Alt
Evangeline Booth Maternity Hospital—W. J. McDonald
Boston City Hospital—Otto J. Hermann
Boston Lying-in Hospital—Frederick C. Irving
Peter Bent Brigham Hospital—Elliott C. Cutler
Robert Breck Brigham Hospital—John G. Kuhns
Carney Hospital—A. M. Fraser
Children's Hospital—Thomas H. Lanman
Corey Hill Hospital—Richard H. Overholt
Faulkner Hospital—E. L. Young
Free Hospital for Women—Frank A. Pemberton
Collis P. Huntington Memorial Hospital—C. C. Simmons
Lakeville State Sanatorium—George W. VanGorder
Malden Hospital—E. J. Reynolds
Massachusetts Eye and Ear Infirmary—F. H. Verhoeff
Massachusetts General Hospital—Arthur W. Allen
Massachusetts Memorial Hospitals—Frank E. Barton
New England Baptist Hospital—N. W. Swinton
New England Deaconess Hospital—B. P. Colcock
New England Hospital for Women and Children—L. D. Adams
New England Medical Center—Samuel H. Proger
Newton Hospital—E. D. Leonard
Palmer Memorial Hospital—Leland S. McKittrick
Pondville Hospital—E. M. Daland
St. Elizabeth's Hospital—Joseph Stanton
Salem Hospital—Walter G. Phippen
United States Marine Hospital—R. L. Waugh
United States Naval Hospital—J. J. A. McMullin

PROGRAMS FOR EVENING SESSIONS

Presidential Meeting and Convocation—Monday, 8:00 p.m.—Symphony Hall

EVARTS A. GRAHAM, M.D., St. Louis; President, American College of Surgeons, Presiding.

Processional—Officers, Regents, and Honorary Guests.

Invocation.

Address of Welcome. LELAND S. MCKITTRICK, M.D., Boston; Chairman, Committee on Arrangements.

Introduction of Foreign Guests. ARTHUR W. ALLEN, M.D., Boston; Vice Chairman, Board of Regents.

Address of Retiring President: American Surgery in a Changing World. EVARTS A. GRAHAM, M.D., St. Louis.

Inauguration of Officers:
President: W. EDWARD GALLIE, M.D., Toronto.
First Vice President: CLARENCE G. TOLAND, M.D., Los Angeles.
Second Vice President: ALBERT C. FURSTENBERG, M.D., Ann Arbor.

Presentation of Initiates for Fellowship. IRVIN ABELL, M.D., Louisville; Chairman, Board of Regents.

Conferring of Fellowships by the President. W. EDWARD GALLIE, M.D., Toronto.

Conferring of Honorary Fellowships. The President.

Medical Records Prize Award.

Tuesday, 8:00 p.m.—Ballroom, Copley-Plaza Hotel

CLARENCE G. TOLAND, M.D., Los Angeles, Vice-President, American College of Surgeons, Presiding.

Annual Oration on Surgery: Some Lessons Learned in the Great War. W. EDWARD GALLIE, M.D., Toronto.

Use of Female Sex Hormones in Clinical Practice. EDWIN C. HAMBLEN, M.D., Durham, N. C.

The Prevention and Treatment of Tetanus. WARFIELD M. FIROR, M.D., Baltimore.

The Care of the Slightly Wounded. WILLIAM DARRACH, M.D., New York.

Wednesday, 8:00 p.m.—Ballroom, Copley-Plaza Hotel

W. EDWARD GALLIE, M.D., Toronto, President, American College of Surgeons, Presiding.

On the Surgical Problems of Modern Warfare. REAR ADMIRAL GORDON GORDON-TAYLOR, Surgeon, Royal Navy, O.B.E., F.R.C.S. (Eng.), London, England.

Endocrine Aspects of Chronic Cystic Mastitis. HOWARD C. TAYLOR, JR., M.D., New York.

Penetrating Wounds of the Heart and Pericardium. R. ARNOLD GRISWOLD, M.D., and C. H. MAGUIRE, M.D., Louisville.

Oration on Fractures and Other Traumas: The General Sugeon's Approach to Problems Presented by Fractures and Other Traumas. WALTER ESTELL LEE, M.D., Philadelphia.

Thursday, 8:00 p.m.—Ballroom, Copley-Plaza Hotel

ARTHUR W. ALLEN, M.D., Boston, Vice-Chairman, Board of Regents, American College of Surgeons, Presiding.

Functional Disturbances of the Hepatic and Choledochous Ducts. PROFESSOR PABLO LUIS MIRIZZI, M.D., Cordoba, Argentina.

Carcinoma of the Stomach (Diagnostic Methods and Therapy). HOWARD K. GRAY, M.D., Rochester, Minn.

Non-penetrating Wounds of the Abdomen. W. L. ESTES, JR., M.D., Bethlehem, Pa.

Adrenal Cortical Tumors; the Types of Non-hormonal and Hormonal Tumors. GEORGE F. CAHILL, M.D., New York.

Evaluation of Blood and Blood Substitutes. ALFRED BLALOCK, M.D., Nashville.

PROGRAMS FOR EVENING SESSIONS

OPHTHALMOLOGY

Tuesday, 8:00 p.m.—Room B, Statler Hotel

EDWIN B. DUNPHY, M.D., Boston, Presiding.

Symposium: Surgery of Heterophoria and Heterotropia.

Preoperative Diagnosis and Treatment. JAMES W. WHITE, M.D., New York.

Trends in the Surgery of Heterotropia. PROFESSOR MOACYR EYCK ALVARO, M.D., Sao Paulo, Brazil.

Surgical Management of Heterophoria. DERRICK T. VAIL, JR., M.D., Cincinnati.

Results of Operation and Causes of Failure. WILLIAM THORNWALL DAVIS, M.D., Washington.

Thursday, 8:00 p.m.—Room B, Statler Hotel

LAWRENCE T. POST, M.D., St. Louis, Member, Advisory Council for Ophthalmology, American College of Surgeons, Presiding.

Symposium: Principles of Ophthalmic Surgery.

Surgical Anatomy of the Eye. F. BRUCE FRALICK, M.D., Ann Arbor, Mich.

Refinements of General Surgical Technique as Applied to Ophthalmology. CONRAD BERENS, M.D., New York.

Management of Surgical Complications. THOMAS D. ALLEN, M.D., Chicago.

OPHTHALMOLOGY AND OTORHINOLARYNGOLOGY

Wednesday, 8:00 p.m.—Room A, Statler Hotel

HARRY S. GRADLE, M.D., Chicago, Regent, American College of Surgeons, Presiding.

Symposium: Neurological Surgery as Related to the Eye and Ear.

Intracranial Infections and Their Spread from the Ear and from the Sinuses. ALBERT C. FURSTENBERG, M.D., Ann Arbor, Mich.

Retrobulbar Neuritis in Relation to Sinus Disease. JAMES B. COSTEN, M.D., St. Louis.

Tumors of the Acoustic Nerve. BYRON STOOKEY, M.D., New York.

Major Surgery of the Orbit. HOWARD C. NAFFZIGER, M.D., San Francisco.

Interpretation of Perimetric Fields of Vision as Manifested in Intracranial Disease. ALBERT D. RUEDEMANN, M.D., CLEVELAND.

OTORHINOLARYNGOLOGY

Tuesday, 8:00 p.m.—Room A, Statler Hotel

ALBERT C. FURSTENBERG, M.D., Ann Arbor, Mich., Vice-President, American College of Surgeons, Presiding.

Sulfonamide Drugs in the Treatment of Suppurative Otitis Media and Mastoiditis. JAMES H. MAXWELL, M.D., Ann Arbor, Mich.

Refinements in the Technique of Laryngectomy. WAITMAN F. ZINN, M.D., Baltimore.

The Otology of Craniocerebral Injuries. WILLIAM E. GROVE, M.D., Milwaukee, Wis.

Thursday, 8:00 p.m.—Room A, Statler Hotel

HARRIS P. MOSHER, M.D., Boston, Presiding.

Symposium: The Salivary Gland.

Benign and Locally Malignant Tumors. ALBERT O. SINGLETON, M.D., Galveston, Texas.

Malignant Tumors. LEROY A. SCHALL, M.D., Boston.

Infections. AUGUST L. BECK, M.D., New Rochelle, N. Y.

PROGRAMS FOR AFTERNOON SESSIONS

CANCER SYMPOSIUM

Tuesday, 2 p.m.—Ballroom, Copley-Plaza Hotel

FRANK E. ADAIR, M.D., New York, Chairman, Cancer Committee, presiding.

Radical Surgical Treatment for Carcinoma of the Cardiac End of the Stomach. JOHN H. GARLOCK, M.D., New York.

Precancerous Lesions. CORNELIUS P. RHOADS, M.D., New York.

Trends in Cancer Research. CARL VOEGTLIN, Ph.D., Bethesda, Md.

Standard Classified Nomenclature of Diseases and Operations, with Especial Reference to Tumors. EDWIN P. JORDAN, M.D., Chicago.

The Rôle of the Radiologist in the Cancer Clinic. GEORGE W. HOLMES, M.D., Boston.

SYMPOSIUM ON FRACTURES AND OTHER TRAUMAS

Wednesday, 2:00 p.m.—Ballroom, Copley-Plaza Hotel

ROBERT H. KENNEDY, M.D., New York; Chairman, Committee on Fractures and Other Traumas, Presiding.

Gunshot Wounds of the Abdomen. AMBROSE H. STORCK, M.D., New Orleans.

A Critical Survey of Ten Years' Experience with Fractures of the Neck of the Femur. MATHER CLEVELAND, M.D., New York.

Factors in the Choice of Material for Bone Plates and Screws. CHARLES S. VENABLE, M.D., San Antonio, Texas.

Treatment of Burns. GROVER C. PENBERTHY, M.D., Detroit.

Results in Cases of Compound Fractures Followed by Osteomyelitis. STANTON K. LIVINGSTON, M.D., Veterans Administration Facility, White River Junction, Vermont.

ASSEMBLY OF INITIATES

Monday, 3:00 p.m.—John Hancock Hall, 90 St. James Avenue

EVARTS A. GRAHAM, M.D., St. Louis, President, Presiding.

Processional—Officers, Regents, and Governors.

Opening Remarks. EVARTS A. GRAHAM, M.D., St. Louis, President.

The Program of the American College of Surgeons. ALBERT O. SINGLETON, M.D., Galveston, Vice President.

The Department of Clinical Research: Cancer; Fractures and Other Traumas; Bone Sarcoma; Hall of the Art and Science of Surgery. BOWMAN C. CROWELL, M.D., Chicago, Associate Director.

Hospital Standardization; Sectional Meetings; Graduate Training in Surgery; Library and Department of Literary Research. MALCOLM T. MACEACHERN, M.D., Chicago, Associate Director.

The Fellowship Pledge. Recital by Initiates.

Greetings to the Initiates. W. EDWARD GALLIE, M.D., Toronto, President-elect.

Fellowship in the College. IRVIN ABELL, M.D., Louisville, Chairman, Board of Regents.

Signing of the Fellowship Roll. The Initiates.

Reception by the Officers and Regents for the Fellows and Initiates and members of their families.

PANEL DISCUSSIONS

Monday, 1:30 to 3:00 p.m.—Statler Hotel

Room B, Mezzanine Floor

Treatment of Acute Suppurative Tenosynovitis of the Fingers, Palm, and Forearm. HUGH AUCHINCLOSS, M.D., New York, Presiding.
Collaborators: WILLIAM E. BROWNE, M.D., Boston; J. H. COUCH, M.D., Toronto; JAMES M. WINFIELD, M.D., Detroit.

Room A, Mezzanine Floor

Vitamins in Surgery. WARREN H. COLE, M.D., Chicago, Presiding.
Collaborators: EMILE HOLMAN, M.D., San Francisco; WALTER ESTELL LEE, M. D., Philadelphia; CHARLES C. LUND, M.D., Boston.

Room C, Mezzanine Floor

Optimum Time for Operation with Special Consideration of Acute Gallbladder Attacks. HENRY F. GRAHAM, M.D., Brooklyn, Presiding.
Collaborators: FRANK GLENN, M.D., New York; CHARLES GORDON HEYD, M.D., New York; E. MACDONALD STANTON, M.D., Schenectady, N. Y.

Georgian Room, Mezzanine Floor

Transfusion and Blood Substitutes, Especially Plasma, in the Prevention and Treatment of Shock. ROY D. MCCLURE, M.D., Detroit, Presiding.
Collaborators: NORMAN E. FREEMAN, M.D., Philadelphia: JOHN SCUDDER, M.D., New York; OWEN H. WANGENSTEEN, M.D., Minneapolis.

Monday, 3:30 to 5:00 p.m.—Statler Hotel

Georgian Room, Mezzanine Floor

The Sulfonamide Group in Abdominal Surgery. FREDERICK A. COLLER, M.D., Ann Arbor, Presiding.
Collaborators: PERRIN H. LONG, M.D., Baltimore; I. S. RAVDIN, M.D., Philadelphia; JAMES E. THOMPSON, JR., M.D., New York.

Room A, Mezzanine Floor

Injuries to the Spine. ERIC OLDBERG, M.D., Chicago, Presiding.
Collaborators: FREDERICK A. JOSTES, M.D., St. Louis; HOWARD C. NAFFZIGER, M.D., San Francisco; JAMES L. POPPEN, M.D., Boston.

Room B, Mezzanine Floor

Thyroid Disease Associated with Congestive Failure. WILLIAM B. PARSONS, M.D., New York, Presiding.
Collaborators: RICHARD B. CATTELL, M.D., Boston; ROBERT S. DINSMORE, M.D., Cleveland; MERRILL N. FOOTE, M.D., Brooklyn.

Room C, Mezzanine Floor

Management of Calcium Urolithiasis. NORMAN W. ROOME, M.D., London, Ont., Presiding.
Collaborators: HENRY O. MERTZ, M.D., Indianapolis; JOHN K. ORMOND, M.D., Detroit; GEORGE G. SMITH, M.D., Brookline, Mass.

Tuesday, 1:30 to 3:00 p.m.—Statler Hotel

Room A, Mezzanine Floor

Advances in Skin Grafting. EARL C. PADGETT, M.D., Kansas City, Mo., Presiding.
Collaborators: JAMES BARRETT BROWN, M.D., St. Louis: MICHAEL L. MASON, M.D., Chicago; HANS MAY, M.D., Philadelphia.

Room B, Mezzanine Floor

Penetrating Abdominal Wounds Caused by Trauma. ARTHUR M. SHIPLEY, M.D., Baltimore, Presiding.
Collaborators: E. CLARENCE MOORE, M.D., Los Angeles; ELKIN L. RIPPY, M.D., Nashville; ALBERT O. SINGLETON, M.D., Galveston.

Georgian Room, Mezzanine Floor

Principles Involved in Hip Joint Arthroplasty. M. N. SMITH-PETERSEN, M.D., Boston, Presiding.
Collaborators: CARL E. BADGLEY, M.D., Ann Arbor, Mich.; GEORGE E. BENNETT, M.D., Baltimore; PHILIP D. WILSON, M.D., New York.

Tuesday, 3:30 to 5:00 p.m.—Statler Hotel

Salle Moderne

Traumas to the Head. FRANCIS C. GRANT, M.D., Philadelphia, Presiding.
Collaborators: CHARLES BAGLEY, JR., M.D., Baltimore; COBB PILCHER, M.D., Nashville; JAMES C. WHITE, M.D., Boston.

Room A, Mezzanine Floor

Treatment of Contaminated and Infected Wounds. FRASER B. GURD, M.D., Montreal, Presiding.
Collaborators: JOSEPH E. HAMILTON, M.D., Louisville; MICHAEL L. MASON, M.D., Chicago; FREDERICK S. WETHERELL, M.D., Syracuse, N. Y.

Room B, Mezzanine Floor

Intestinal Obstruction in Children. WILLIAM E. LADD, M.D., Boston, Presiding.
Collaborators: WILLIS D. GATCH, M.D., Indianapolis; GEORGE B. PACKARD, M.D., Denver; GROVER C. PENBERTHY, M.D., Detroit.

Georgian Room, Mezzanine Floor

Gastric Ulcer. URBAN MAES, M.D., New Orleans, Presiding.
Collaborators: RALPH COLP, M.D., New York; VERNE C. HUNT, M.D., Los Angeles; GEORGE P. MULLER, M.D., Philadelphia.

Wednesday, 1:30 to 3:00 p.m.—Statler Hotel

Room B, Mezzanine Floor

Graduate Training in Surgery in Hospitals (First Session). ROBIN C. BUERKI, M.D., Philadelphia, Presiding.
Collaborators: DAVID H. BALLON, M.D., Montreal; GEORGE F. CAHILL, M.D., New York; EDWARD D. CHURCHILL, M.D., Boston; JOHN R. FRASER, M.D., Montreal; W. EDWARD GALLIE, M.D., Toronto; HARRY S. GRADLE, M.D., Chicago; DALLAS B. PHEMISTER, M.D., Chicago; STANLEY J. SEEGER, M.D., Milwaukee, Wis.; PHILIP D. WILSON, M.D., New York.

Room C, Mezzanine Floor

Management of Abortion. CONRAD G. COLLINS, M.D., New Orleans, Presiding.
Collaborators: RALPH E. CAMPBELL, M.D., Madison, Wis.; JOHN R. FRASER, M.D., Montreal; H. CLOSE HESSELTINE, M.D., Chicago.

Room A, Mezzanine Floor

Developments in the Field of Poliomyelitis Research. EDWARD L. COMPERE, M.D., Chicago, Presiding.
Collaborators: FREMONT A. CHANDLER, M.D., Chicago; CHARLES L. LOWMAN, M.D., Los Angeles; OSCAR L. MILLER, M.D., Charlotte, N. C.

Georgian Room, Mezzanine Floor

Lung Surgery. STUART W. HARRINGTON, M.D., Rochester, Minn., Presiding.
Collaborators: RICHARD H. MEADE, JR., M.D., Philadelphia; HAROLD NEUHOF, M.D., New York; WILLIAM F. RIENHOFF, JR., M.D., Baltimore.

Wednesday, 3:30 to 5:00 p.m.—Statler Hotel

Room B, Mezzanine Floor

Graduate Training in Surgery in Hospitals (Second Session). ROBIN C. BUERKI, M.D., Philadelphia, Presiding.
Collaborators: DAVID H. BALLON, M.D., Montreal; GEORGE F. CAHILL, M.D., New York; EDWARD D. CHURCHILL, M.D., Boston; JOHN R. FRASER, M.D., Montreal; W. EDWARD GALLIE, M.D., Toronto; HARRY S. GRADLE, M.D., Chicago; DALLAS B. PHEMISTER, M.D., Chicago; STANLEY J. SEEGER, M.D., Milwaukee, Wis.; PHILIP D. WILSON, M.D., New York.

Georgian Room, Mezzanine Floor

Postoperative Pulmonary Complications. LEO ELOESSER, M.D., San Francisco, Presiding.
Collaborators: HENRY K. BEECHER, M.D., Boston; STUART W. HARRINGTON, M.D., Rochester, Minn.; RICHMOND L. MOORE, M.D., New York.

Room A, Mezzanine Floor

Disruption of Abdominal Wounds. FRANK GLENN, M.D., New York, Presiding.
Collaborators: LAWRENCE S. FALLIS, M.D., Detroit; L. KRAEER FERGUSON, M.D., Philadelphia; HILGER P. JENKINS, M.D., Chicago.

Room C, Mezzanine Floor

Continuous Spinal Anesthesia. WILLIAM T. LEMMON, M.D., Philadelphia, Presiding.
Collaborators: H. H. BRADSHAW, M.D., Winston-Salem, N. C.; RALPH T. KNIGHT, M.D., Minneapolis; EDWARD B. TUOHY, M.D., Rochester, Minn.

Thursday, 3:30 to 5:00 p.m.—Statler Hotel

Room C, Mezzanine Floor

Treatment of Chronic Cervicitis. GEORGE H. GARDNER, M.D., Chicago, Presiding.
Collaborators: JOHN C. BURCH, M.D., Nashville; J. MASON HUNDLEY, JR., M.D., Baltimore; JOE V. MEIGS, M.D., Boston.

Room A, Mezzanine Floor

Chemotherapy in Wound Healing. EDWARD L. HOWES, M.D., New York, Presiding.
Collaborators: HILGER P. JENKINS, M.D., Chicago; FRANK L. MELENEY, M.D., New York; J. ROSS VEAL, M.D., Washington.

Georgian Room, Mezzanine Floor

The Treatment of the Wound in Compound Fractures. CLAY RAY MURRAY, M.D., New York, Presiding.
Collaborators: R. ARNOLD GRISWOLD, M.D., Louisville; J. ALBERT KEY, M.D., St. Louis; HENRY C. MARBLE, M.D., Boston.

Room B, Mezzanine Floor

Anesthesia for Surgical Procedures within the Upper Abdomen. RALPH M. TOVELL, M.D., Hartford, Conn., Presiding.
Collaborators: HENRY K. BEECHER, M.D., Boston; HOWARD K. GRAY, M.D., Rochester, Minn.; HENRY S. RUTH, M.D., Merion, Pa.

Salle Moderne

Contributions to Medical Progress through Postmortem Examinations. ALAN R. MORITZ, M.D., Boston, Presiding.
Collaborators: A. O. GETTLER, Ph.D., New York; HARRISON S. MARTLAND, M.D., Newark, N. J.

Friday, 1:30 to 3:00 p.m.—Statler Hotel

Room B, Mezzanine Floor

Chemotherapy in Genitourinary Infections or in Infections of the Prostatic Gland. EDWIN P. ALYEA, M.D., Durham, N. C., Presiding.
Collaborators: GEORGE F. CAHILL, M.D., New York; GEORGE C. PRATHER, M.D., Boston.

Room C, Mezzanine Floor

Diseases of the Esophagus. ALTON OCHSNER, M.D., New Orleans, Presiding.
Collaborators: CARL EGGERS, M.D., New York; FRANK H. LAHEY, M.D., Boston; GABRIEL TUCKER, M.D., Philadelphia.

Georgian Room, Mezzanine Floor

The Management of the Perforated Appendix, Early and Late. HENRY K. RANSOM, M.D., Ann Arbor, Mich., Presiding.
Collaborators: J. MONTGOMERY DEAVER, M.D., Philadelphia, CLARENCE E. GARDNER, JR., M.D., Durham, N. C.; E. ERIC LARSON, M.D., Los Angeles.

Room A, Mezzanine Floor

Experience with the Miller-Abbott Tube. ALLEN O. WHIPPLE, M.D., New York, Presiding.
Collaborators: WILLIAM O. ABBOTT, M.D., Philadelphia; CHARLES G. JOHNSTON, M.D., Detroit; ANGUS MCLACHLIN, M.D., Toronto.

Friday, 3:30 to 5:00 p.m.—Statler Hotel

Room B, Mezzanine Floor

Fractures of the Facial Bones. LLOYD NOLAND, M.D., Fairfield, Ala., Presiding.
Collaborators: ROBERT H. IVY, M.D., Philadelphia; CARL W. WALDRON, M.D., Minneapolis; CHARLES H. WILSON, M.D., New Orleans.

Georgian Room, Mezzanine Floor

Surgical Diseases of the Colon. CHARLES W. MAYO, M.D., Rochester, Minn., Presiding.
Collaborators: THOMAS G. ORR, M.D., Kansas City, Mo.; HOWARD PATTERSON, M.D., New York; J. WILLIAM THOMPSON, M.D., St. Louis.

GROUP CLINICAL CONFERENCES

OPHTHALMOLOGY AND OTORHINOLARYNGOLOGY

Tuesday, 11:00 a.m.—Statler Hotel

Surgery of Squint. JOHN H. DUNNINGTON, M.D., New York, Presiding.
Treatment of Sinus Disease in Children. SAMUEL SALINGER, M.D., Chicago, Presiding.

Wednesday, 11:00 a.m.—Statler Hotel

Surgery of the Tear Passages. WALTER S. ATKINSON, M.D., Watertown, N. Y., Presiding.
Indications for Surgery of the Nose and Throat in Diabetics. WESTLEY M. HUNT, M.D., New York, Presiding.

Thursday, 11:00 a.m.—Statler Hotel

Surgery of the Orbit. ALGERNON B. REESE, M.D., New York, Presiding.
Surgical Treatment of Deafness. SAMUEL J. KOPETZKY, M.D., New York, Presiding.

ANNUAL MEETING, BOARD OF GOVERNORS

Wednesday, 12:00 m.—Salon, Copley-Plaza Hotel

W. EDWARD GALLIE, M.D., Toronto; President, American College of Surgeons, Presiding.

Statement by the Chairman of the Board of Regents. IRVIN ABELL, M.D., Louisville.

Types of Problems Encountered by the Administrative Board and the Central Committee on Credentials and Methods of Dealing with Them: FREDERIC A. BESLEY, M.D., Waukegan, Secretary; MALCOLM T. MACEACHERN, M.D., Chicago, Chairman, Administrative Board; BOWMAN C. CROWELL, M.D., Chicago, Vice Chairman, Administrative Board.

Discussion by the Governors and Regents.

ADJOURNED MEETING, BOARD OF GOVERNORS

Thursday, 1:30 p.m.—Ballroom, Copley-Plaza Hotel

Report of Committee on Nominations to the Board of Governors.

ANNUAL MEETING, FELLOWS OF THE COLLEGE

Thursday, 1:45 p.m.—Ballroom, Copley-Plaza Hotel

W. EDWARD GALLIE, M.D., Toronto; President, American College of Surgeons, Presiding.

Report of Committee on Nominations to the Fellows.

Library and Department of Literary Research. MISS L. MARGUERIETE PRIME, Chicago; Librarian.

Financial Report. DALLAS B. PHEMISTER, M.D., Chicago; Treasurer.

Departmental Reports: (a) Hospital Standardization. (b) Work of Credentials Committees, Committees on Applicants, and Committee on History Reviews. (c) State and Provincial Sectional Meetings. (d) Medical Motion Pictures. (e) Public Relations. MALCOLM T. MACEACHERN, M.D., Chicago; Associate Director.

Graduate Training in Surgery—General Surgery and the Surgical Specialties. DALLAS B. PHEMISTER, M.D., Chicago; Chairman, Committee on Graduate Training in Surgery.

Hall of the Art and Science of Surgery. GEORGE CRILE, M.D., Cleveland; Chairman, Committee on the Hall of the Art and Science of Surgery.

Committee on Cancer. FRANK E. ADAIR, M.D., New York; Chairman.

Committee on Fractures and Other Traumas. ROBERT H. KENNEDY, M.D., New York; Chairman.

Department of Clinical Research. BOWMAN C. CROWELL, M.D., Chicago; Associate Director.

College Administration. FREDERIC A. BESLEY, M.D., Waukegan; Secretary.

Fellowship Obligations and Opportunities. ARTHUR W. ALLEN, M.D., Boston; Vice Chairman, Board of Regents.

The College in Its Relation to National Defense. IRVIN ABELL, M.D., Louisville; Chairman, Board of Regents.

COMMITTEE MEETINGS

IRVIN ABELL, M.D., Louisville; Chairman, Board of Regents, Presiding.

STATE AND PROVINCIAL JUDICIARY COMMITTEES

Wednesday, 9:30 a.m.—John Hancock Hall, 90 St. James Avenue

Statement by the Chairman of the Board of Regents.
Selection and appointment of judiciary committees.
Procedure in dealing with judiciary cases.
Types of cases referred to the judiciary committees.
Discussion by Fellows and Regents of the College.

STATE AND PROVINCIAL EXECUTIVE COMMITTEES

Wednesday, 10:00 a.m.—John Hancock Hall, 90 St. James Avenue

Statement by the Chairman of the Board of Regents.
Sectional Meetings Held in 1941 and Plans for Sectional Meetings in 1942.
Comments on Sectional Meetings in 1941, by the Chairmen of the Committees on Local Arrangements:

1. Minneapolis, Minnesota. WILLIAM A. HANSON, M.D.
2. Pittsburgh, Pennsylvania. HOLLAND H. DONALDSON, M.D.
3. Salt Lake City, Utah. RALPH T. RICHARDS, M.D.

Discussion by the Fellows and Regents of the College.

CREDENTIALS COMMITTEES AND COMMITTEES ON APPLICANTS

Wednesday, 10:30 a.m.—John Hancock Hall, 90 St. James Avenue

Statement by the Chairman of the Board of Regents.
Summary of Work of Credentials Committees and Committees on Applicants, 1941.
Demonstration—Interviewing a Candidate for Fellowship by a Committee on Applicants. CHARLES S. KENNEDY, M.D., Detroit, Chairman; ELMER R. ARN, M.D., Dayton; FRANK K. BOLAND, M.D., Atlanta; VERNE C. HUNT, M.D., Los Angeles; FREDERIC V. HUSSEY, M.D., Providence; CHARLES B. PUESTOW, M.D., Chicago; GEORGE K. RHODES, M.D., San Francisco.
The Junior Candidate. JAMES E. THOMPSON, M.D., New York.
Discussion by the Fellows and Regents of the College.

FORUM ON FUNDAMENTAL SURGICAL PROBLEMS

Tuesday, Wednesday, Thursday, Friday, 9:30 a. m.—Ballroom, Copley-Plaza Hotel

OWEN H. WANGENSTEEN, M.D., Minneapolis, Chairman of Committee, Presiding.
Presentation of the results of clinical and experimental research on problems related to general surgery and the surgical specialties being currently conducted by the departments of surgery in many of our medical schools. Detailed programs for these sessions will be published in the official program and the *Daily Clinical Bulletin*.

ANNUAL HOSPITAL STANDARDIZATION CONFERENCE

Monday, 10:00 a.m.—Ballroom, Copley-Plaza Hotel

EVARTS A. GRAHAM, M.D., St. Louis; President, American College of Surgeons, presiding.

Address of the President—The Hospital Program of the American College of Surgeons.

Report of the 1941 Hospital Standardization Survey—Official Announcement of the List of Approved Hospitals. IRVIN ABELL, M.D., Louisville; Chairman, Board of Regents, and EARLE W. WILLIAMSON, M.D., Chicago; Assistant Director, American College of Surgeons.

Report on Graduate Training in Surgery. DALLAS B. PHEMISTER, M.D., Chicago; Chairman, Committee on Graduate Training in Surgery, and HAROLD EARNHEART, M.D., Chicago; Assistant Director, American College of Surgeons.

Important Legislative, Preparedness and Emergency Problems Now Facing Hospitals. BERT W. CALDWELL, M.D., Chicago; Executive Secretary, American Hospital Association.

The Preservation of Our Voluntary System of Hospitals. REV. ALPHONSE M. SCHWITALLA, S.J., St. Louis; President, Catholic Hospital Association.

The Responsibility of the Medical Staff in Maintaining Control and Quality of the Professional Work in the Hospital. MERRILL N. FOOTE, M.D., Brooklyn.

National Events and Their Effect on Personnel Relations. FRANK J. WALTER, Denver.

Hospital Preparedness in the Defense Program. JAMES A. HAMILTON, New Haven, Conn.

Monday, 2:00 p.m.—Ballroom, Copley-Plaza Hotel

Panel Round Table Conference: Meeting the Problems of Rendering Adequate Care of the Patient and Maintaining Quality Standards of Service During the Present Period of Preparedness and National Emergency. NATHANIEL W. FAXON, M.D., Boston, presiding.

Discussion from the standpoint of professional personnel:
Medical Staff. NORBERT A. WILHELM, M.D., Boston.
Resident Medical Staff. CLAUDE W. MUNGER, M.D., New York.
Nursing Staff. SALLY JOHNSON, R.N., Boston.

Discussion from the standpoint of medical, surgical and hospital equipment, instruments and supplies:
Scientific Equipment and Instruments. D. H. PALMER, New York.
Surgical Dressings, Rubber Goods and Other Supplies. L. M. ARROWSMITH, Brooklyn.
Surgical Ligatures, Pharmaceuticals and Chemicals. EVERETT W. JONES, Albany, N. Y.

Discussion from the standpoint of operating costs:
Foodstuffs, Supplies of All Kinds, Commodities, etc. MIRIAM CURTIS, R.N., Syracuse, N.Y.
Salaries and Wages. ABRAHAM OSEROFF, Pittsburgh.
Supplementary Revenue: Subsidies, Gifts, Donations, Endowments, etc. OLIVER H. BARTINE, Bridgeport, Conn.

General Discussion. Other related subjects may also be presented if time permits.

Monday, 4:30 p.m.—Foyer, Copley-Plaza Hotel

Consultation Service. Opportunity will be afforded everyone attending the Hospital Standardization Conference to consult with recognized leaders in the hospital field, on any question or problem on which specific information is desired. A consultation service will be available at the time and place specified in the program, by means of pre-arranged information stations, on six different phases of hospital service daily. This service will be provided by leaders in the various fields of hospital administration and management.

Administrative Practices and Policies. EDGAR C. HAYHOW, Paterson, N. J.

Anesthesia Service. E. B. TUOHY, M.D., Rochester, Minn.

Clinical Laboratory and Pathological Service. WILMAR M. ALLEN, M.D., Hartford, Conn.

Control of Major Surgery in the Hospital. E. MACDONALD STANTON, M.D., Schenectady, N.Y.

Food Service in the Hospital—Diet Therapy. GENEVA MARBLE, Boston.

Plans for Hospitals in the Present Emergency. OLIVER G. PRATT, Salem, Mass.

Tuesday, 7:45 a.m.—Salon, Copley-Plaza Hotel

Breakfast Conference, Sponsored by the American College of Hospital Administrators in co-operation with the American College of Surgeons: Forum on Fundamental Problems of Hospital Administration. SCOTT WHITCHER, New Bedford, Mass., presiding.

Discussion from the following aspects:
Relation of Personal Qualities and Attitudes to Successful Hospital Administration. G. HARVEY AGNEW, M.D., Toronto, Ont.
Critical Analysis of Present Methods of Training for Hospital Administration. E. M. BLUESTONE, M.D., New York.

General Discussion. Opened by GERHARD HARTMAN, Chicago.

Tuesday, 9:30 a.m.—Swiss Room, Copley-Plaza Hotel

Panel Discussion: Essentials Pertaining to an Efficient Obstetrical Department in the Hospital. HARVEY B. MATTHEWS, M.D., Brooklyn leader. Collaborators: JOHN R. FRASER, M.D., Montreal; JOSEPH L. BAER, M.D., Chicago; JAMES R. MILLER, M.D., Hartford; CLARA M. KONRAD, R.N., Jersey City, N.J.

Topics for discussion: Accommodations, facilities and equipment of a modern obstetrical department. Supervision and management of the obstetrical department. Personnel—resident, medical and nursing staffs. Qualifications of the medical staff in the practice of obstetrics within the hospital. Control of the clinical work. Essentials of a complete obstetrical record. Control of infections and complications. Periodic analytical summaries of end results. Care of the newborn. Graduate training in obstetrics.

Tuesday, 9:30 a.m.—Sheraton Room, Copley-Plaza Hotel

Panel Discussion: Essentials Pertaining to an Efficient Department of Orthopedic Surgery in the Hospital. HENRY H. KESSLER, M.D., Newark, N. J., leader. Collaborators: FRANK D. DICKSON, M.D., Kansas City, Mo.; FREMONT A. CHANDLER, M.D., Chicago; JOELLE C. HIEBERT, M.D., Lewiston, Maine; WILLIAM B. SELTZER, New York.

Topics for discussion: Accommodations, facilities and equipment of a department of orthopedic surgery in a hospital. Supervision and management of the department of orthopedic surgery. Personnel—resident, medical and nursing staffs. Qualifications of the medical staff to do orthopedic surgery. Control of the

clinical work of the department of orthopedic surgery. Essential contents of the medical record in orthopedic surgery. Review and analysis of the clinical work of the department of orthopedic surgery. Splint and brace shop. Follow-up and study of end-results. Graduate training in orthopedic surgery.

Tuesday, 11:00 a.m.—Swiss Room, Copley-Plaza Hotel

Panel Discussion: Organization and Functioning of the Medical Staff. JOSEPH C. DOANE, M.D., Philadelphia, leader. Collaborators: STANLEY J. SEEGER, M.D., Milwaukee; JULIUS H. COMROE, York, Pa.; CHARLES B. PUESTOW, M.D., Chicago; ALLAN CRAIG, M.D., Bangor, Maine; MERRILL N. FOOTE, M.D., Brooklyn.

Topics for discussion: Standards of qualifications in making appointments to the medical staff. Procedure in making appointments to the medical staff. Legal responsibility of the hospital in respect to the professional work of the hospital. Supervision and control of major surgery and major obstetrics. Basic considerations in making promotions on the medical staff of the hospital. Essential features in conducting the medical staff conference. Maintaining adequate medical records. Increasing the number of consultations in hospital practice. Supervision of the work of the resident medical staff. Responsibility of the senior members of the medical staff in instructing junior members.

Tuesday, 11:00 a.m.—Sheraton Room, Copley-Plaza Hotel

Panel Discussion: Essentials Pertaining to an Efficient Department of Anesthesia in the Hospital. RALPH T. KNIGHT, M.D., Minneapolis, leader. Collaborators: RALPH M. TOVELL, M.D., Hartford; HENRY RUTH, M.D., Merion, Pa.; ELEANOR F. HOWARD, M.D., Boston.

Topics for discussion: Accommodations, facilities and equipment of the department of anesthesia in the hospital. Supervision and management of the department of anesthesia. Essential qualifications for the efficient administration of anesthesia. Anesthesia records to be maintained. A critical analysis of newer types of anesthetics and methods of administration. Hazards associated with the administration of anesthetics. Training of interns and residents in the administration of anesthetics. Follow-up and study of end-results in the administration of anesthetics. Handling of emergency anesthetics. Graduate training in anesthesiology.

Tuesday, 2:00 p.m.—Swiss Room, Copley-Plaza Hotel

WILSON W. KNOWLTON, M.D., Boston, presiding.

The Organization of a Blood Transfusion Department in a General Hospital. LEO M. ZIMMERMAN, M.D., and SIDNEY O. LEVINSON, M.D., Chicago.

Organization and Use of the Medical Library by the Medical Staff and Personnel of the Hospital. L. MARGUERITE PRIME, Chicago.

A Successful Plan in Handling the Personnel Health Service in the Hospital. JOSEPH G. NORBY, Milwaukee.

The Control and Assignment of Duties of Non-Professional Workers in the Hospital. FRANCES C. LADD, R.N., Boston.

Tuesday, 4:30 p.m.—Foyer, Copley-Plaza Hotel

Consultation Service:

Internships and Residencies. M. G. WESTMORELAND, M.D., Chicago.

Medical Record Problems. EDNA K. HUFFMAN, R.R.L., Chicago.

Medical Social Service. MABEL R. WILSON, Boston.

Medical Staff Problems. PAUL S. FERGUSON, M.D., Chicago.

Sterilization of Surgical Dressings, Instruments, Rubber Goods, Supplies, etc. SAMUEL R. D. HEWITT, M.D., St. John, New Brunswick.

Tuesday, 8:00 p.m.—Swiss Room, Copley-Plaza Hotel

Panel Discussion: The Hospital Trustee in his Relationships and Responsibilities. RAYMOND P. SLOAN, New York, leader. Collaborators: FRANK O. ROBSON, Southbridge, Mass.; JOHN MACGREGOR, Boston; SAMUEL STEWART, Lewiston, Maine; CHARLES F. WILINSKY, M.D., Boston.

Topics for discussion: Qualifications of the ideal hospital trustee. Acquiring essential knowledge concerning hospital administration. Relationship of the hospital trustee to (a) the administration; (b) the medical staff. The problem of the hospital trustee in selecting a hospital administrator. The trustee's responsibility in the increasing financial responsibility of the hospital. The hospital trustee's approach to problems with possible legal or liability significance. Meeting the responsibility of the present demands on hospitals incident to the preparedness program. Preparation and conduct of the monthly meeting of the board of trustees. Monthly and annual reports. The trustee's participation in public relations of the hospital.

Motion picture: White Battalions—Serving All Mankind.

Wednesday, 7:45 a.m.—Salon, Copley-Plaza Hotel

Breakfast Conference, Sponsored by the Boston Medical Record Librarians Association in co-operation with the American College of Surgeons: Forum on Fundamental Considerations in the Art and Science of Medical Record Keeping. W. FRANKLIN WOOD, M.D., Waverley, Mass., presiding.

Discussion from the following aspects:

A Critical Analysis of the Present Ways and Means of Training Medical Record Librarians. SISTER M. LORETTA, R.R.L., Duluth, Minn.

The Place of the Medical Record Librarian in the Hospital Organization. MARGARET DUBOIS, M.D., Chicago.

What the Medical Record Librarian Can Contribute Towards Making a Success of Her Career. MARGARET TAYLOR, R.R.L., Rochester, N. Y.

Wednesday, 9:30 a.m.—Swiss Room, Copley-Plaza Hotel

Joint Conference with American Association of Medical Record Librarians. ROBIN C. BUERKI, M.D., Philadelphia, presiding.

Our Challenge—Good Medical Records in Our Hospitals. ANNA C. SCHULZE, R.R.L., Philadelphia.

The Rôle of the Record Librarian in the Changing World. SISTER MARY SERVATIA, R.R.L., St. Louis.

The Preparation of Medical Statistics and Special Studies for the Medical Staff. MARY M. NEWTON, R.R.L., Evanston, Ill.

Medical Photography as an Aid to More Comprehensive Medical Records. C. G. BROWNELL, Rochester, N. Y.

Organization and System in Handling Tumor Clinic Records. FLORENCE G. BABCOCK, R.R.L., Ann Arbor, Mich.

The Introduction and Orientation of the Intern to Medical Record Keeping in the Hospital. MARSHALL DAVISON, M.D., Chicago.

Round Table Conference—Discussion of the following topics: The filming of medical records. The present status of the standard nomenclature. Recording of infections in the medical record. The place of the medical secretary in securing medical records. Floor supervision of current

medical records. The use of the dictaphone in securing medical records. Assisting the members of the medical staff by providing statistical and other information for scientific presentations. Co-operation between the medical record department and the medical library. Ideal setup for the medical record room. Postgraduate courses for medical record librarians.

Wednesday, 2:30 p.m.—Local Hospitals

GROUP CONFERENCES AND DEMONSTRATIONS

Beth Israel Hospital. CHARLES F. WILINSKY, M.D., Director.
- Administration, Business Methods, Hospital Accounting, and Other Activities. SAMUEL D. KAUFMAN.
- Admitting and Discharging Procedures. NATALIE H. NATHAN.
- Operating Room Organization and Management; Setup for Major Operation—Supplies, Facilities, and Personnel. MARY C. GILMORE, R.N.
- Organization and Management of a Blood Bank. BENJAMIN ALEXANDER, M.D.

Boston Dispensary. (New England Medical Center). FRANK E. WING, Director.
- Out-patient Department—Organization, Clinic Management and Control, Admitting, and Other Administrative Procedures. EDITH R. CANTERBURY and KATHARINE E. WAKEFIELD.
- Pharmacy Service—Organization, Management, and Service to Out-patients. HAROLD E. SANDBERG.
- Health Education in an Out-Patient Department. FRANCES STERN and MARY PFAFFMAN.
- Purchasing, Accounting, and Central Store Supply. ABBIE E. DUNKS.

Boston Lying-in Hospital. WILSON W. KNOWLTON, M.D., Superintendent.
- Study Tour of Hospital; General Routine; Technique of Isolation of Obstetrical Patients; Identification of Infants; Care of the Premature; Technique for Maintenance of Breast Feeding; Directory for Mothers' Milk; Other Features of General Interest. GERTRUDE GARRAN, R.N.

Children's Hospital. GEORGE VON L. MEYER, Director.
- Administration of Blood and Plasma Transfusions to Infants. House officer and graduate nurse.
- Nursing Care of the Infant Surgical Patient. Nursing supervisor.
- The New Arthur T. Legg Room for Functional Rehabilitation of the Handicapped; Pool and Underwater Exercises; Different Methods of Physiotherapy for Children. Physiotherapy Staff of Children's Hospital and Harvard Infantile Paralysis Committee.

Haynes Memorial Hospital, Brookline (Massachusetts Memorial Hospitals). HENRY M. POLLOCK, M.D., Superintendent.
- Study Tour of Contagious Hospital—Technique in the Care of Communicable Diseases. DR. SMITH.

Joseph H. Pratt Diagnostic Hospital (New England Medical Center). SAMUEL PROGER, M.D., Medical Director.
- Graduate Teaching in Medicine, Dietetics and Laboratory Methods. SAMUEL PROGER, M.D.
- Clinical Laboratory—Organization, Management, and Service. JOSEPH BENOTTI, M.S.

Massachusetts Eye and Ear Infirmary. NATHANIEL W. FAXON, M.D., Director.
- Nursing Technique for Eye, Ear, and Throat Conditions. MARIE SCHERER, R.N.
- Bronchoscopic Clinic—Organization, Management of Patients, and Technical Procedures. JESSIE STEWART, R.N.

Massachusetts General Hospital. NATHANIEL W. FAXON, M.D., Director.
- Medical Records; Organization, Management, and Service; Complete Unit Record System, Soundex System of Filing Cards; Follow-up and End Results. GENEVIEVE C. CHASE.
- Anesthesia Department: Organization, Management, and Service. HENRY K. BEECHER, M.D.
- Diagnostic Clinic: A Consultative Service for Private Patients. T. STEWART HAMILTON, M.D.

Massachusetts Memorial Hospitals. HENRY M. POLLOCK, M.D., Superintendent.
- Nursery Management; Mothers' Milk Supply; Procedure and Technique in Making Infants' Formulas. MISS HODGON.
- Organization and Management of a Blood Bank. DR. BARTON.
- Anesthesia Department—Organization, Management, and Service. DR. FERGUSON.
- Electrocardiographic Activities in a General Hospital. JAMES M. FAULKNER, M.D.

New England Baptist Hospital. GEORGIE M. BOULTER, Acting Superintendent.
- Operating Room Organization and Management; Setup for Major Operation—Facilities, Supplies, Personnel. ARDIS RAUPACH.
- Air-conditioning in Operating Suite. WALTER E. LOVERING and ARDIS RAUPACH.
- Special Diet—Building Special Diets on House Menu. HELEN G. MCDONALD.
- Gift Shop. BESSIE A. WHITE.

New England Deaconess Hospital. REV. WARREN F. COOK, Superintendent.
- Nursing Procedure with Neurosurgical Patients. MARJORIE B. DAVIS, R.N.
- Conducting a Profitable Pharmacy Store. HOWARD PRINGLE.
- Social Service Procedures with Diabetic and Cancer Patients. GERTRUDE S. JAMESON.

Peter Bent Brigham Hospital. NORBERT A. WILHELM, M.D., Superintendent.
- Preparation of Parenteral Solutions—Technique of Administering. CARL W. WALTER, M.D.
- Sterilization of Supplies and Instruments. CARL W. WALTER, M.D.

Wednesday, 4:30 p.m.—Foyer, Copley-Plaza Hotel

Consultation Service:
- Nursing Service. MARY SHEPARD, R.N., Cambridge, Mass.
- Obstetrical Department Management. FRANCES YOUNG, Boston.
- Occupational Therapy. LUCY G. MORSE, O.T.R., Boston.
- Outpatient Department Problems. A. G. ENGELBACH, M.D., Cambridge, Mass., and SHERMAN THORNDIKE, M.D., Boston.
- Personnel Management and Personnel Relations. FRANK J. WALTER, Denver, Colo.
- Physical Therapy. MARY NESBITT, Boston.
- Public Relations. JOSEPH P. LEONE, M.D., Quincy, Mass.

Wednesday, 8:00 p.m.—Swiss Room, Copley-Plaza Hotel

Motion Pictures for Hospital Personnel: How We Hear; How We See (sound); Foods and Nutrition (sound); Heart and Circulation (sound); A New Day (deals with pneumonia) (sound); Goodbye, Mr. Germ; Proper Care and Use of Hypodermic Needles; White Battalions—Serving All Mankind.

Thursday, 7:45 a.m.—Salon, Copley-Plaza Hotel

Breakfast Conference on Hospital Public Relations.

Discussion from the following aspects:

Underlying Principles of the Public Relations Program. Prof. MAX R. GROSSMAN, Boston.

The Written Word—Newspaper Publicity. WESLEY FULLER, Boston.

The Spoken Word—Radio Talks. DEL CASTILLO, Boston.

The Visual Way—Tours Through the Hospital and Motion Pictures.

Thursday, 9:30 a.m.—Swiss Room, Copley-Plaza Hotel

Panel Discussion: Essentials Pertaining to an Efficient Department of Urology in the Hospital. GEORGE F. CAHILL, M.D., New York, leader. Collaborators: LEO P. DOLAN, M.D., Toledo; GILBERT J. THOMAS, M.D., Minneapolis; GEORGE O'HANLON, M.D., Jersey City, N.J.

Topics for discussion: Accommodations, facilities and equipment of a department of urology in the hospital. Supervision and management of the department of urology. Personnel management—resident, medical and nursing staffs. Qualifications of the medical staff to do urology. Control of the clinical work of the department of urology. Essentials of the medical record in urology. Regular periodic review and analysis of the clinical work in urology. Follow-up and study of end results. Graduate training in urologic surgery.

Thursday, 11:00 a.m.—Swiss Room, Copley-Plaza Hotel

Panel Discussion: Essentials Pertaining to an Efficient Department of Ophthalmology and Otolaryngology in the Hospital. CONRAD BERENS, M.D., and MARVIN F. JONES, M.D., New York, leaders. Collaborator: LAWRENCE T. POST, M.D., St. Louis.

Topics for discussion: Accommodations, facilities and equipment of the department of ophthalmology and otolaryngology. Supervision and management of the department of ophthalmology and otolaryngology. Personnel of the department of ophthalmology and otolaryngology—resident, medical and nursing staffs. Qualifications of the medical staff to do ophthalmological and otolaryngological surgery. Control of the clinical work in the department of ophthalmology and otolaryngology. Regular periodic review and analysis of the clinical work of the ophthalmological and otolaryngological department. Essentials of the medical record for ophthalmological and otolaryngological patients. Anesthetic service for the ophthalmological and otolaryngological patients. Training of interns and nurses in the ophthalmological and otolaryngological department. Graduate training in ophthalmology and otolaryngology.

Thursday, 2:00 p.m.—Swiss Room, Copley-Plaza Hotel

Round Table Conference: Administrative practices, policies, personnel; business methods; medical staff organization; medical staff conferences; medical records; diagnostic and therapeutic services; obstetrical, nursing, food and pharmacy services; outpatient department; housekeeping; maintenance and utilities; hospital auxiliaries; and other practical problems. ROBIN C. BUERKI, M.D., Philadelphia, and G. HARVEY AGNEW, M.D., Toronto, Ont., leaders.

Collaborators: GEORGE O'HANLON, M.D., Jersey City, N. J., SIDNEY G. DAVIDSON, New Haven, Conn.; LEWIS E. JARRETT, M.D., Richmond, Va.; E. MURIEL MCKEE, R.N., Brantford, Ont.; SISTER MARY ALPHONSUS, Teaneck, N. J.; EDNA H. NELSON, R.N., Chicago; SISTER M. PATRICIA, O.S.B., Duluth, Minn.; EDNA D. PRICE, R.N., Concord, Mass.; L. V. RAGSDALE, M.D., Grand Rapids, Mich.; HELEN ROBINSON, R.N., Wauseon, Ohio; DAN TRANER, Lynn, Mass.; FRANK J. WALTER, Denver.

PRELIMINARY CLINICAL PROGRAM

GENERAL SURGERY

Monday

BOSTON CITY HOSPITAL

Staff—2. Symposium: Vascular disease.
S. J. G. NOWAK—2. Experimental hypertension.
E. A. EDWARDS—2:30. Thrombophlebitis in unusual forms.
E. E. O'NEIL—3. Lumbar sympathectomy for vascular disease.
D. C. GOLDFARB—3:30. False aneurysm of femoral artery.
T. LEARY—4. Pathological aspects of vascular disease.
E. E. O'NEIL—4:30. Refrigeration in gangrene of lower extremities.

COLLIS P. HUNTINGTON MEMORIAL HOSPITAL

Staff—2:15. Dry clinic:
JOSEPH C. AUB. Recent advances in our knowledge of bone diseases.
CHANNING C. SIMMONS. Malignant tumors of bone.
RICHARD DRESSER. Radiographic demonstration of bone tumors and certain rare forms of skeletal disease.
CHARLES E. DUNLAP. Carcinogenic agents.
CLIFFORD C. FRANSEEN. Industrial cancer.

MASSACHUSETTS GENERAL HOSPITAL

H. K. BEECHER and associates—2. Dry clinic: Anesthesia and related subjects.
H. K. BEECHER—2. Use of ether in the presence of pulmonary tuberculosis; anesthesia for transpleural gastric surgery.
JULIA G. ARROWOOD—2:25. Fractional spinal anesthesia.
H. K. BEECHER—2:35. Anesthesia and shock.
Staff—3:10 Discussion.
EDWARD B. BENEDICT—3:30. Endoscopy, bronchoscopy and esophagoscopy with special emphasis on the two newer endoscopic methods, gastroscopy and peritoneoscopy; demonstration with lantern slides and patients.
Staff—2. Symposium: Peripheral vascular disease; varicose veins and their complications.
H. H. FAXON—2. Fundamental considerations, anatomy, physiology, etiology, examination.
R. H. THOMPSON—2:15. Late results following treatment of uncomplicated varicose veins.
H. H. FAXON—2:30. Present concept of treatment.
R. R. LINTON—2:45. Chronic and recurrent ulceration of lower extremities, etiological factors and classification; present concept of treatment.
Staff—3:30. Discussion.
H. H. FAXON, R. H. THOMPSON, and R. R. LINTON—4. Operations: Peripheral vascular.
E. M. DALAND, A. O. HAMPTON, and associates—2. Dry clinic: Tumors.
A. O. HAMPTON—2. Roentgen diagnosis of cancer of the lung.
G. W. HOLMES—2:15. Roentgen treatment of cancer of the lung.
J. V. MEIGS—2:30. Changing concepts in the treatment of cancer of the cervix.
E. M. DALAND—2:45. The locus of recurrence in cancer of the breast.
R. SCHATZKI—3. End-results of roentgen therapy in cancer of the larynx.
L. L. ROBBINS—3:15. The treatment of polycythemia vera by spray radiation.
T. B. MALLORY—3:30. Malignant ulcers of the stomach.
C. E. WELCH—3:45. Gastric ulcers in relation to gastric cancer.
J. R. LINGLEY—4. Comparison of skin effect on the albino rat of 200 k.v. and 1200 k.v. roentgen ray.
G. W. TAYLOR—4:15. The value of regional dissection in cancer cases.

NEW ENGLAND BAPTIST HOSPITAL

H. D. ADAMS and N. W. SWINTON—1:30. Operative clinic: Thyroid, stomach, colon, rectum and biliary tract.
U. H. EVERSOLE, LEO V. HAND, and M. J. NICHOLSON—1:30. Demonstrations: Anesthesia.

NEW ENGLAND DEACONESS HOSPITAL

H. D. ADAMS and N. W. SWINTON—1:30. Operative clinic: Thyroid, stomach, colon, rectum, and biliary tract.
U. H. EVERSOLE, LEO V. HAND, and M. J. NICHOLSON—1:30. Dry clinic: Anesthesia in thyroid surgery, including intratracheal anesthesia, pre-operative and post-operative medication, recent developments in spinal anesthesia.
CARL SOUDERS. Postoperative pulmonary complications.
JAMES EVANS. Thrombophlebitis and pulmonary embolism.

NEW ENGLAND MEDICAL CENTER

(Joseph H. Pratt Diagnostic Hospital)

Staff—2. Dry clinic:
JOSEPH H. PRATT—2. Value of secretin test in diagnosis of pancreatic disease.
WILLIAM DAMESHEK—2:30. Indications for splenectomy; case reports.
JOHN D. ADAMS—3. Various conditions found in the hip joint following injury.
EDWARD L. YOUNG—3:30. Peritoneoscopy.

PALMER MEMORIAL HOSPITAL

Staff—2. Symposium: Carcinoma of the breast.
CLIFFORD C. FRANSEEN. End-results of surgical treatment.
L. S. MCKITTRICK. Interstitial irradiation treatment of carcinoma of the breast with platinum radium needles.
J. H. MARKS. X-ray therapy of carcinoma of the breast.
SHIELDS WARREN. Relation between chronic cystic mastitis and carcinoma of the breast.

Tuesday

BETH ISRAEL HOSPITAL

Staff—9. Dry clinic:
ARNOLD STARR. Biliary tract disease, left-sided pain.
C. G. MIXTER. Technique of immediate cholangiography; motion pictures.
L. A. HERMANSON. Cholangiography, indications for and end-results.
H. A. FRANK. Advantages of intravenous synthetic vitamin K_1 over other vitamin K preparations.
A. M. SELIGMAN. Vitamin P in capillary fragility.
Staff—10:30. Operative clinic:
C. G. MIXTER. Cholecystectomy and cholangiography.

JACOB FINE. Cholecystectomy, transverse incision.
L. H. NASON. Peritoneoscopy, spinal anesthesia.
ARNOLD STARR. Peritoneoscopy, local anesthesia.
Staff—2. Dry clinic:
MAX DAVIS. Ovarian tumors with endocrine effects.
R. B. DAVIDOFF. Relationship of cystic disease of the breast to disease of the uterus and adnexa.
L. A. HERMANSON. Comparative merits of the vaginal smear and endometrial biopsy in diagnosis of ovarian function.
S. L. CARGILL. Hyperparathyroidism.
D. D. BERLIN. Carcinoma of the thyroid.
M. F. LESSES. Complications of thyroid surgery.
S. L. CARGILL. Management of recurrent and persistent thyrotoxicosis.
D. D. BERLIN. The avoidance of complications in thyroid surgery.

Scientific Exhibit—Daily

JOSEPH GOODMAN. Pathogenic fungi in foot infections.
H. F. FRIEDMAN. Malignant disease before and after treatment by radiation.
ABRAHAM RUDY and S. R. MUELLNER. Neurogenic bladder in diabetes mellitus.
JULIUS LOMAN. Action of autonomic drugs on the gastro-intestinal and genito-urinary tracts.
S. A. ROBINS. Urethrography and cystography.
W. S. ALTMAN and L. A. HERMANSON. Cholangiography.
LEONARD NATHAN and KURT THOMA. Cysts and tumors of mouth and jaw.
JULIUS LOMAN. Cerebral arteriography.
ARNOLD STARR and H. A. FRANK. Venography in deep phlebitis of lower leg.
S. S. HANFLIG. Mechanical device for reduction of Colles' fracture.

BOSTON CITY HOSPITAL

Staff—2. Dry clinic: Gastric lesions.
W. R. MORRISON—2. Gastrojejunocolonic fistula.
C. W. MCCLURE—2:30. Gastroscopy, kodachrome pictures.
A. R. KIMPTON—3. Cancer of stomach.
W. R. MORRISON—3:30. Perforated peptic ulcer.
E. E. O'NEIL—4. Transpleural approach for carcinoma of upper end of stomach.
T. W. WICKHAM—4:30. Cholecystitis and its relation to peptic ulcer. Review of six hundred cholecystectomies.

PETER BENT BRIGHAM HOSPITAL

ELLIOTT C. CUTLER and ROBERT E. GROSS—9. Operations: Ligation of patent ductus arteriosus; pericardiectomy.
ELLIOTT C. CUTLER and associates—10. Symposium: Surgery of the heart and blood vessels.
C. SIDNEY BURWELL. Forms of heart disease amenable to surgery.
SAMUEL A. LEVINE. Cardiac disease simulating surgical conditions of the abdomen and vice versa.
EUGENE C. EPPINGER. Indications for surgery for patent ductus arteriosus.
ROBERT E. GROSS. Surgical treatment of patent ductus arteriosus.
ELLIOTT C. CUTLER. The technique of pericardiectomy.
MERRILL C. SOSMAN. The rôle of the roentgenologist in cardiac surgery.
JOHN HOMANS. Peripheral vascular disease.
MARSHALL N. FULTON. Angioma of skeletal muscle.
DAVID CHEEVER—2. Demonstration: Surgical anatomy of the abdomen.

CHILDREN'S HOSPITAL

WILLIAM E. LADD and associates—10:30. Dry clinic: Diseases of the gastro-intestinal tract in childhood, followed by questions and discussion.
WILLIAM E. LADD. Atresia, stenosis, and malrotation.
THOMAS H. LANMAN. Pyloric stenosis.
ROBERT E. GROSS. Intussusception.
HENRY W. HUDSON, JR. Appendicitis.

MASSACHUSETTS GENERAL HOSPITAL

A. W. ALLEN and associates—8. Symposium: Gastric lesions.
C. M. JONES—8. Medical considerations.
A. O. HAMPTON and associates—8:30. Roentgenological studies.
E. B. BENEDICT—8:50. Gastroscopy and peritoneoscopy.
C. E. WELCH—9:10. Gastric ulcer.
LANGDON PARSONS—9:30. Cancer of the stomach.
R. H. SWEET—9:50. Transthoracic gastrectomy.
A. W. ALLEN—10:10. Surgical procedures for gastric lesions.
Staff—10:30. Operations: Gastric resections for benign and malignant lesions.
H. H. FAXON, HORATIO ROGERS, and associates—9. Symposium: Acute appendicitis.
HORATIO ROGERS. Present status; chief factors in present mortality rates, late diagnosis and/or treatment, faulty appraisal of individual patients, rigid policies of treatment, faulty technique, type of incision.
W. D. SMITH. Appendicitis in general medical practice.
HORATIO ROGERS. Appendiceal peritonitis before localization.
H. H. FAXON. Appendiceal peritonitis after localization.
CHAMP LYONS. The rôle of bacteriology and chemotherapy in appendicitis.
R. R. LINTON. Special aspects of appendicitis in young children.
Staff—11. Operative clinic.
L. S. MCKITTRICK and associates—2. Symposium: Surgery of the colon and rectum.
L. S. MCKITTRICK—2. Operations for carcinoma of the rectum and their indications.
A. W. ALLEN—2:20. The management of carcinoma of the colon.
E. P. HAYDEN—2:40. The complications of diverticulitis of the sigmoid and their management.
ROY E. MABREY—3. The operative and non-operative management of internal hemorrhoids.
Staff—3:20. Discussion.
Staff—3:40. Operative clinic.
CHAMP LYONS and FRANK L. MELENEY (New York)—2. Dry clinic: Surgical infections.

MASSACHUSETTS MEMORIAL HOSPITALS

HOWARD M. CLUTE, CLIFFORD D. HARVEY, CHARLES SZIKLAS, FRANK E. BARTON, and HOLLIS L. ALBRIGHT—8. Operative clinic: Thyroid, gall-bladder, stomach, and colon.
ELEANOR FERGUSON, REGINALD HUNT, and M. JEAN BLACK. Demonstrations: Anesthesia.
Staff—10. Symposium: Management of blood-bank.
FRANK E. BARTON. Management of a blood-bank in a small hospital.
CHARLES BRANCH. Correlation of the hospital laboratory with the blood-bank.
JOHN SCUDDER (New York). Studies in treatment of shock with plasma and serum.
CORNELIUS P. RHOADS (New York). Present application and future development of blood substitute therapy.

PHILLIPS L. BOYD—10. Dry clinic: Lymphogranuloma venereum with rectal involvement.
FRANK E. BARTON—2. Demonstration of transfusion service.
Staff—2. Symposium: Surgery of the biliary tract.
KNOWLES LAWRENCE—2. Intrinsic disease of the liver simulating cholecystitis.
HOLLIS L. ALBRIGHT—2:20. Non-calculous gall bladder.
THOMAS J. ANGLEM—2:40. Complications of common duct surgery.
FRANZ INGLEFINGER—3. Duodenal drainage in diagnosis of biliary tract disease.
CHESTER KEEFER—3:20. Liver abscess as a cause of prolonged fever.
HOWARD M. CLUTE—3:40. Stricture of the bile ducts.

NEW ENGLAND BAPTIST HOSPITAL

F. H. LAHEY, R. B. CATTELL, S. F. MARSHALL, and B. P. COLCOCK—9. Operative clinic: Thyroid, stomach, colon, and biliary tract.
U. H. EVERSOLE, LEO V. HAND, and M. J. NICHOLSON—9. Anesthesia demonstrations: Regional, spinal, continuous spinal, and general.
H. D. ADAMS and N. W. SWINTON—1:30. Operative clinic: Thyroid, stomach, colon, rectum, and biliary tract.
U. H. EVERSOLE, LEO V. HAND, and M. J. NICHOLSON—1:30. Demonstrations: Anesthesia.

NEW ENGLAND DEACONESS HOSPITAL

F. H. LAHEY, R. B. CATTELL, S. F. MARSHALL, and B. P. COLCOCK—9. Operative clinic: Thyroid, stomach, colon, and biliary tract.
U. H. EVERSOLE, LEO V. HAND, and M. J. NICHOLSON—9. Anesthesia demonstrations: Regional, spinal, continuous spinal, and general.
H. D. ADAMS and N. W. SWINTON—1:30. Operative clinic: Thyroid, stomach, colon, rectum, and biliary tract.
U. H. EVERSOLE, LEO V. HAND, and M. J. NICHOLSON—1:30. Demonstrations: Anesthesia.
F. H. LAHEY, LEWIS M. HURXTHAL, ELMER C. BARTELS, and H. J. PERKINS—2. Symposium: Thyroid surgery: Subtotal thyroidectomy; diagnosis of hyperthyroidism; diagnosis and treatment of the thyrocardiac; complications following thyroid surgery; recent developments in blood iodine studies.

NEW ENGLAND HOSPITAL FOR WOMEN AND CHILDREN

ELEANOR E. COWAN—10. Out-patient clinics: Gynecological, medical, and pediatric.
G. LINDH MULLER—10. Pathological presentation: Carcinoid tumor of small intestine, metastases to liver; carcinoma of gastro-intestinal tract.
RACHEL L. HARDWICK, BIANCA R. LIA, and MERRY E. PITTMAN—2. Dry clinic: Correlation of electrostethocardiography with roentgenography in rheumatic heart disease, congenital heart disease, and functional abnormalities, with presentation of cases.

NEW ENGLAND MEDICAL CENTER
(Joseph H. Pratt Diagnostic Hospital)

Staff—2. Dry clinic:
EDWARD T. WHITNEY—2. Bleeding per ano, illustrated with lantern slides.
C. H. LAWRENCE—2:30. Masculinizing factors in the female.
K. S. ANDREWS, HENRY H. LERNER, and WILLIAM M. SHEDDEN—3:30. Recent advances in diagnosis and treatment of cancer of the stomach.

PONDVILLE HOSPITAL

E. M. DALAND, G. W. TAYLOR, H. ROGERS, T. J. ANGLEM, R. H. WALLACE, and associates—9. Operative clinic: Cancer.
Staff—11. Dry clinic:
E. M. DALAND. End-results in cancer of the breast.
T. J. ANGLEM. Cancer of the mouth, demonstration of cases.
G. W. TAYLOR. Lymph node metastases.
S. WARREN and associates. Pathological demonstration.
C. E. DUMAS and associates. Roentgen demonstration of bone tumors.

ST. ELIZABETH'S HOSPITAL

Staff—9. Operative and dry clinic:
JOSEPH STANTON—9. Thyroidectomy.
MICHAEL E. MCGARTY—9. Bilateral recurrent inguinal hernia.
JOSEPH STANTON—10. Biliary tract operation.
FRANCIS JANTZEN—10:30. Discussion of sub-deltoid bursitis and ruptured supraspinatus tendon.
WILLIAM F. DOLAN—11. Wrist injuries in industry.
MICHAEL E. MCGARTY—11:30. Skin grafts, demonstration of cases.

SALEM HOSPITAL

W. G. PHIPPEN, DONALD A. NICKERSON, and S. A. WILSON—10. Dry clinic: Uses and abuses of laboratory and roentgenological procedures in abdominal surgery.
E. L. PEIRSON—11. Prostatic surgery with some newer methods of diagnostic approach.

UNITED STATES MARINE HOSPITAL

RICHEY L. WAUGH and associates—8. Operative clinic: Hernioplasty, indirect, direct, and femoral; appendectomy; ligation of saphenous vein; hemorrhoidectomy.
RICHEY L. WAUGH and associates—1. Dry clinic:
JAMES F. SPINDLER. Parasitic appendicitis.
KENNETH HAUSFELD. Femoral hernia, a simple operation with report of cases.
RICHEY L. WAUGH. Terminal ileitis.
JOHN A. PRIOR. Rupture of spleen, delayed hemorrhage.

UNITED STATES NAVAL HOSPITAL

J. J. A. MCMULLIN and staff—9. Operations.

Wednesday

BETH ISRAEL HOSPITAL

Staff—9. Dry clinic:
L. H. NASON. Pressure treatment of keloids.
J. B. SEARS. Prophylaxis of pulmonary embolism by division of femoral vein.
EDWARD HIRSCH. Changes in volume of the peritoneal cavity following intercostal nerve paralysis.
C. G. MIXTER. Giant hernia.
ARNOLD STARR. Advanced regional ileitis.
C. G. MIXTER. Transverse ileocolostomy with or without resection for regional ileitis.
Staff—10:30. Operative clinic:
C. G. MIXTER. Intestinal resection.
J. B. SEARS. Division of femoral vein for treatment of non-fatal pulmonary embolus.
D. D. BERLIN. Thyroidectomy.
Staff—2. Dry clinic:
M. D. ALTSCHULE. Obstruction of superior vena cava.
BENJAMIN ALEXANDER. Blood bank.
MAX DAVIS and SAMUEL GILMAN. Venous pressure changes in lower extremities during and after abdominal surgery.

S. B. BEASER. Rare earth anticoagulants.
JACOB FINE. Thrombophlebitis and embolism, diagnostic and therapeutic considerations.
BENJAMIN ALEXANDER. Polycythemia vera in peripheral vascular disease.
B. F. MASSELL and S. B. BEASER. Evaluation of therapy of peripheral vascular disease.
JACOB FINE. Adrenal cortical hormones in shock and hemorrhage.

BOSTON CITY HOSPITAL

Staff—9. Symposium: Cancer.
Department of Pathology—9. Splenogenic myeloid metaplasia; argentophil tumors (carcinoid).
DR. JOLIFF—9:30. Surgical treatment of myeloma.
F. W. O'BRIEN—9:50. Cancer and x-ray therapy.
E. A. COONEY—10:20. Carcinoma of breast.
A. R. KIMPTON—10:45. Cancer of large intestine.
CHARLES C. LUND—11:10. Cancer of mouth.
WILLIAM MORRISON—11:35. Cancer of stomach.
Staff—2. Symposium: Thyroid disease.
R. C. COCHRANE—2. Tumors of parathyroid, lantern slides.
D. D. BERLIN—2:30. Hyperthyroidism in cancer of thyroid.
W. T. SALTER—2:50. Graves' disease without hypertension.
RICHARD SMITH—3:10. Adenoma of thyroid.
Staff—2. Dry clinic:
GORDON M. MORRISON—2. Radial nerve injury with fracture of humerus.
J. EDWARD FLYNN—2:30. Anatomical and clinical investigations of deep fascial space abscess of hand.
T. K. RICHARDS—3:15. Traumatic injuries of the knee joint.
A. P. AITKEN—3:45. Epiphyseal separation.
ROBERT ULIN—4:30. Clubfeet.
Staff—3:30. Symposium: Vitamins in surgery.
CHARLES C. LUND and J. H. CRANDON—3:30. Vitamin C and wound healing.
WALTER WEGNER and MERRILL MOORE—4:15. Vitamins in presence of head injuries.
DR. MIKELJOHN—4:45. Presentation of cases.

PETER BENT BRIGHAM HOSPITAL

ELLIOTT C. CUTLER and ROBERT ZOLLINGER—9. Operations: Gastric resections.
ELLIOTT C. CUTLER and associates—10. Symposium: Gastric and duodenal ulcer.
ELLIOTT C. CUTLER. Indications for surgery.
EDWARD S. EMERY, JR. Medical management of bleeding peptic ulcer.
MERRILL C. SOSMAN. Significance of prepyloric ulcer.
HARRY A. WARREN. Duodenal diverticulum.
ROBERT ZOLLINGER. Surgical treatment of severe duodenal ulcer.
ELLIOTT C. CUTLER. Results of acute gastroduodenal perforation.
CARL W. WALTER—2. Symposium: Aseptic technique.

CARNEY HOSPITAL

A. MCK. FRASER, WILLIAM E. BROWNE, and staff—9. Operative clinic: Gastro-intestinal, biliary tract, thyroid and hand.
Staff—2. Dry clinic:
WILLIAM E. BROWNE. Moving picture demonstration and brief description of unusual lesions of arm, forearm, and hand, showing condition present before treatment and end-results. Cases will include xanthomata, glomus tumor, so-called trigger finger contractures, aneurysms in hand, melanotic carcinoma, tendon suture repair with transplanted segment of vein for covering at site of anastomosis, difference between real and pseudo-neuromata in amputation stumps, ulnar nerve repair, series of median nerve cases, results in a series of cases following radical operation for relief of Dupuytren's contracture with 90 to 100 per cent return of normal function of the hand.
DAVID C. DITMORE. Vital tissue staining, its practical application in office and hospital proctology, with report of results in a series of 1000 private patients.
W. R. MACAUSLAND. Fracture and dislocation of carpal-semilunar bones.
HAROLD G. LEE. End-results in lumbosacral fusion; end-results of epicondylar fractures of humerus.
A. LEO BRETT. Case presentation of myeloma of 12 years' duration with complete absorption of tenth vertebra.
JOHN L. DOHERTY. A simple method of treating fractures of surgical neck of humerus.
WILLIAM C. MALONEY. Practical application of clinical laboratory procedures in jaundice.
A. MCK. FRASER. End-results of gall-bladder surgery.

CHILDREN'S HOSPITAL

WILLIAM E. LADD and associates—9. Operations.

DILLON FIELD HOUSE

(Soldiers' Field, Allston)

AUGUSTUS THORNDIKE, JR., and associates—3. Dry clinic: Athletic injuries. Summary of recent developments in the physiology of physical fatigue.

FAULKNER HOSPITAL

Staff—8. Operative and dry clinic:
E. L. YOUNG, A. R. KIMPTON, F. G. BALCH, H. K. SOWLES, R. F. SHELDON, E. E. O'NEIL, R. S. TITUS, and R. J. HEFFERNAN. Operations.
S. C. WIGGIN. Anesthesia exhibits.
J. B. HAZARD. Pathological specimens, gross and microscopic.
Staff—2. Operative and dry clinic:
A. R. KIMPTON—2. Gastric resection.
H. K. SOWLES—2:15. Reconstruction operation for hypertrophied breast.
E. L. YOUNG—2:30. Stricture of papilla of vater.
R. C. COCHRANE—2:45. Parathyroid tumors.
WM. C. QUINBY—3. Importance of water balance in diseases of the prostate.
E. E. O'NEIL—3:15. Prevention of pulmonary embolism by ligation and division of femoral vein.
R. F. SHELDON—3:30. Local infiltration anesthesia for pelvic delivery.
R. J. HEFFERNAN—3:45. Use of progesterin in treatment of habitual and threatened abortion.
DR. MALONEY—4. Management of bleeding in jaundice.
R. S. TITUS—4:15. Diabetes in pregnancy.
DR. STRAUSS—4:30. Importance of vitamins.
S. C. WIGGIN—4:45. Anesthesia for complicated surgical cases.

MALDEN HOSPITAL

LOUIS E. PHANEUF—9. Operation: Vaginal hysterectomy.
IRVING J. WALKER—9. Operation: Cholecystectomy.
Staff—10. Dry clinic and demonstrations:
GORDON D. ATKINSON—10. Urology.
RUSSELL F. SULLIVAN—11. Orthopedic surgery.
LOUIS E. PHANEUF—11:30. Gynecology.
IRVING J. WALKER—12. Ludwig's angina.
J. S. ROONEY—12:30. Pathology.
KENNETH K. DAY—1. Blood bank.

Staff—2. Operative clinic:
GORDON D. ATKINSON—2. Nephrectomy.
N. A. GALLAGHER—2:30. Hysterectomy.
RUSSELL F. SULLIVAN—3:30. Spinal fusion (Hibbs').

MASSACHUSETTS GENERAL HOSPITAL

L. S. MCKITTRICK and associates—9. Operative and dry clinic: Ulcerative colitis.
C. M. JONES—9. Medical aspects, diagnosis and treatment.
L. S. MCKITTRICK—9:20. Surgical aspects, types of operations and indications.
J. D. STEWART—9:40. Ileostomy, operative technique, postoperative complications.
RICHARD WARREN—10:10. Late-results after ileostomy, social and economic aspects.
L. S. MCKITTRICK—10:30. Colectomy, indications, technique and results.
Staff—10:45. Discussion.
Staff—11. Operations.
H. C. MARBLE and associates—9. Dry clinic: Surgical conditions of the hand.
H. C. MARBLE. Anatomy and examination.
F. G. BALCH, JR. Hand tumors.
EDWARD HAMLIN, JR. Injuries to nerves of upper extremity.
BRADFORD CANNON. Skin grafts of hand.
A. L. WATKINS. Physiotherapy in reconstruction.
A. W. REGGIO. Occupational therapy in reconstruction.
H. C. MARBLE. Surgical reconstruction.
A. W. ALLEN and associates—2. Symposium: Diseases of the gall bladder and bile ducts.
A. W. ALLEN—2. Technical considerations of the gall bladder and bile ducts with special reference to morbidity and mortality.
RICHARD WALLACE—2:30. Acute cholecystitis.
J. D. STEWART—2:50. Obstructive jaundice, liver function, preparation of the patient and after-care.
RICHARD WARREN—3:15. Carcinoma of the gall bladder.
Staff—3:30. Operative clinic: Extrahepatic biliary tract.
Staff—2. Symposium: Peripheral vascular clinic; obliterative arterial disease.
L. S. MCKITTRICK—2. Fundamental considerations, classification, differential diagnosis.
EDWARD HAMLIN, JR.—2:15. Thrombo-angiitis obliterans: Outline of treatment, nerve crushing, mechanical aids, indications for amputation.
H. E. KENNARD—2:35. Thrombo-angiitis obliterans: End-result study with particular reference to the economic aspect of the disease.
RICHARD WARREN—2:55. Arteriosclerotic gangrene with and without diabetes, indications for and technique of minor amputation.
T. C. PRATT—3:10. Arteriosclerotic gangrene with and without diabetes, indications for and technique of supracondylar amputation.
R. H. SMITHWICK—3:30. Lumbar sympathectomy, its rôle in the treatment of obliterative disease; indications for operation and operative technique.
Staff—3:50. Discussion.
Staff—4. Operative clinic: Supracondylar amputation for gangrene, nerve crushing for relief of pain, lumbar sympathectomy.

MASSACHUSETTS MEMORIAL HOSPITALS

HOWARD M. CLUTE, CLIFFORD D. HARVEY, CHARLES SZIKLAS, FRANK E. BARTON, and HOLLIS L. ALBRIGHT—8. Operative clinic: Thyroid, gall bladder, stomach, and colon.
ELEANOR FERGUSON, REGINALD HUNT, and M. JEAN BLACK. Demonstrations: Anesthesia.
GEORGE LEVENE and EGON G. WISSING—9:30. Demonstration: X-ray examination of the rectum and sigmoid with special reference to lateral view.
Staff—10. Symposium: Gastric surgery.
HOWARD M. CLUTE. Gastric hemorrhage.
JOHN SPRAGUE. End-result studies after gastroduodenostomy.
FRANZ INGLEFINGER. Miller-Abbott tube in gastrointestinal diagnosis.
THOMAS J. ANGLEM. Cancer of the stomach.
ELEANOR FERGUSON. Anesthesia for upper abdominal surgery.
FRANK E. BARTON—2. Demonstration of transfusion service.

NEW ENGLAND BAPTIST HOSPITAL

F. H. LAHEY, R. B. CATTELL, S. F. MARSHALL, and B. P. COLCOCK—9. Operative clinic: Thyroid, stomach, colon, and biliary tract.
U. H. EVERSOLE, LEO V. HAND, and M. J. NICHOLSON—9. Anesthesia demonstrations: Regional, spinal, continuous spinal, and general.
H. D. ADAMS and N. W. SWINTON—1:30. Operative clinic: Thyroid, stomach, colon, rectum, and biliary tract.
U. H. EVERSOLE, LEO V. HAND, and M. J. NICHOLSON—1:30. Demonstrations: Anesthesia.

NEW ENGLAND DEACONESS HOSPITAL

F. H. LAHEY, R. B. CATTELL, S. F. MARSHALL, and B. P. COLCOCK—9. Operative clinic: Thyroid, stomach, colon, and biliary tract.
U. H. EVERSOLE, LEO V. HAND, and M. J. NICHOLSON—9. Anesthesia demonstrations: Regional, spinal, continuous spinal, and general.
H. D. ADAMS and N. W. SWINTON—1:30. Operative clinic: Thyroid, stomach, colon, rectum, and biliary tract.
U. H. EVERSOLE, LEO V. HAND, and M. J. NICHOLSON—1:30. Demonstrations: Anesthesia.
R. B. CATTELL, N. W. SWINTON, and EVERETT D. KEIFER—2. Dry clinic: Diagnosis and treatment of carcinoma of rectum; rectal surgery for benign lesions; diagnosis and treatment of ulcerative colitis.
JOHN NORCROSS. Avitaminosis and recent developments in intravenous use of heperin.

NEW ENGLAND HOSPITAL FOR WOMEN AND CHILDREN

Staff—10. Out-patient clinics: Gynecological, skin and allergy, pediatric and well-baby.
G. LINDH MULLER—10. Pathological demonstration: Breast cancer.

NEW ENGLAND MEDICAL CENTER
(Joseph H. Pratt Diagnostic Hospital)

Staff—9. Dry clinic:
RICHARD H. OVERHOLT—9. The operability of primary carcinoma of the lung.
REEVE H. BETTS—9:30. The management of acute pulmonary abscess.
H. E. MACMAHON—10. Gastric lesions in malignant hypertension.
S. J. THANNHAUSER—10:30. Liver function tests as an aid to gall-bladder surgery.

NEWTON HOSPITAL

Staff—9. Operative clinics.
G. KENNETH COONSE—9. A new arthroplasty of the hip.
LOUIS PILCHER—9. Appendectomy.

R. I. SMITH—9:30. Thyroidectomy for hyperthyroidism.
M. P. BRACKETT—9:30. Cholecystectomy.
D. G. NUTTER—10. Hysterectomy for fibroids.
E. D. LEONARD—10. Breast amputation.
HERBERT DUNPHY—11. Skin graft.
Staff—2. Dry clinic.
G. K. COONSE. Diagnosis and treatment of shoulder injuries.
R. I. SMITH. Tumors of the neck.
G. D. NUTTER. New apparatus for fractures of the clavicle.
CHARLES LAMB. Peripheral vascular clinic.
H. G. DUNPHY. Management of cicatricial contractures due to burns, skin grafting in general.
E. D. LEONARD. Arterial thrombosis in a young girl.

ST. ELIZABETH'S HOSPITAL

Staff—9. Operative and dry clinics:
JOHN W. SPELLMAN—9. Surgery of the breast.
JOSEPH STANTON—9. Gastric surgery.
EDWARD M. HODGKINS—9. Herniorrhaphy, fascial strips.
LAWRENCE J. LOUIS—10. Cholecystectomy.
JOHN G. DOWNING—11. Surgical complications due to fungi. Colored motion picture, "A Clinical and Laboratory Study of Fungus Diseases."
JOHN W. SPELLMAN—11. Treatment of advanced cancer, demonstration of cases.
WILLIAM NOONAN—11:45. Spinal anesthesia.

UNITED STATES NAVAL HOSPITAL

LAWRENCE J. MCCARTHY and staff—9. Operations.
JOHN R. MARSHALL and CHARLES H. SWAN—9. Dry clinic: Circulatory diseases of the extremities, case histories.

Thursday

BETH ISRAEL HOSPITAL

Staff—9. Dry clinic:
S. S. HANFLIG. Use of traction in treatment of cervical arthritis.
MYER KARP. Treatment of acute subdeltoid bursitis by needling, novocain and irrigation; mechanics of fracture of surgical neck of humerus, methods of reduction.
M. H. ROGERS. Manipulation for chronic adhesive bursitis.
ARMIN KLEIN. Chemotherapy of acute sepsis of bones and joints.
W. S. ALTMAN. Delayed roentgenological evidence of fracture.
ARMIN KLEIN. Intervertebral disc, orthopedic aspects.
W. JASON MIXTER. Intervertebral disc, neurological and surgical aspects.
DAVID AYMAN. Results of sympathectomy for hypertension.
Staff—10:30. Operative clinic:
R. H. SMITHWICK. Sympathectomy for hypertension.
W. JASON MIXTER. Removal of intervertebral disc.
M. H. ROGERS. Arthrotomy of shoulder joint.
Staff—2. Dry clinic:
ALFRED HURWITZ. Sulfanilamide vs. zinc peroxide in the treatment of phagedenic ulcer.
LOUIS ZETZEL. The Miller-Abbott tube.
M. D. ALTSCHULE. Effect of abdominal surgery on respiratory dynamics.
H. L. BLUMGART. The surgical risk in cardiac patients.
JACOB FINE. Treatment of intestinal distention by sulfaguanidine.
ARNOLD STARR. Sulfaguanidine for the sterilization of isolated loops of colon.

BEVERLY HOSPITAL

Staff—2. Dry clinic:
PEER P. JOHNSON. Acute pancreatic disease, its diagnosis and treatment.
BARNARD P. TODD. Adenoma of the islets of Langerhans.
RICHARD E. ALT. Abdominal manifestations of renal disease.
PEER P. JOHNSON and HAMMOND J. DUGAN. Results of deferred operation in acute perforated appendicitis.
JOSEPH A. CUNNINGHAM. A clinicopathological study of abdominal Hodgkin's disease.

BOSTON CITY HOSPITAL

OTTO J. HERMANN and associates—9. Symposium: Low back pain.
OTTO J. HERMANN—9. Introduction.
JOHN T. WILLIAMS—9. Gynecological aspects.
AUGUSTUS RILEY—9:15. Genito-urinary aspects.
ROBERT ULIN—9:30. A rational method of routine back examination based on anatomical and physiological factors.
JOSEPH SHORTELL—9:45. Low back pain with negative x-ray, lumbosacral; sacro-iliac myositis; myositis fascitis, industrial postural, mechanical findings.
THOMAS H. PETERSON—10. Coccygodynia fracture of coccyx, conservation vs. surgery in treatment.
RUSSELL SULLIVAN—10:15. Anatomical abnormalities of fifth lumbar vertebra and lumbosacral joint.
G. KENNETH COONSE—10:30. Spondylolisthesis, pathology, diagnostic points, rôle of trauma in its production and treatment.
A. P. AITKEN—10:45. Fascial contractions.
MARK H. ROGERS—11. Intervertebral disc problem, orthopedic aspect.
P. F. BUTLER—11:15. Intervertebral disc problem, x-ray aspect.
DONALD MUNRO—11:30. Intervertebral disc problem, neurological aspect.
Staff—2. Operative and dry clinic: Appendicitis, biliary system, and hernia.
P. S. FOISIE—2. Appendicitis and complications.
G. W. PAPEN—2:30. Incision for appendectomy in extreme obesity.
F. F. HENDERSON and JOHN MCGOWAN—3. Operative biliary drainage for relief of jaundice, operative care of gall-bladder patient.
J. J. HEPBURN—3:20. Familial hemolytic jaundice and splenectomy.
W. R. MORRISON—3:40. Stones of the hepatic ducts subdiaphragmatic abscess.
CHARLES LAMB—4. Peritoneoscopy with special reference to biliary disease.
RICHARD SMITH—4:20. Exteriorization of small intestines in strangulated hernia.
D. C. GOLDFARB—4:40. Recurrent inguinal hernia.
Staff—2. Symposium: Anesthesia, and abdominal surgery.
T. W. WICKHAM—2. Foci of infection and ulcerative colitis.
A. R. KIMPTON—2:15. Surgery of large intestine.
C. W. MCCLURE—2:30. Sigmoidoscopy, kodachrome pictures.
FRANK MARVIN—2:45. Indications and contra-indications for various anesthetics.
S. C. WIGGIN—3. Spinal anesthesia.
G. C. MOORE—3:15. Intravenous anesthesia.
P. S. FOISIE—3:30. Ruptured corpus hemorrhagicum.
ALLEN DAVIS—3:45. Calcified cyst of spleen.
S. J. MADDOCK—4. High and low intestinal obstruction.
ALLEN DAVIS—4:20. Gun shot wounds of abdomen.
HALSEY B. LODER—4:40. Postoperative treatment.

PETER BENT BRIGHAM HOSPITAL

FRANCIS C. NEWTON and J. ENGLEBERT DUNPHY—9. Operations: Colon, rectum and anus.

ELLIOTT C. CUTLER and associates—10. Symposium: Colon, rectum and anus.

FRANCIS C. NEWTON. Experiences with cancer of the colon.

J. ENGLEBERT DUNPHY. Experiences with anterior resection for cancer of the rectosigmoid.

ANDREW W. CONTRATTO and THOMAS B. QUIGLEY. Differential diagnosis of gastritis, gastro-enteritis and appendicitis.

MERRILL C. SOSMAN. The x-ray and large bowel surgery.

EDWARD S. EMERY, JR. Medical diseases of the colon.

ELLIOTT C. CUTLER. Cicatrizing enteritis.

CARNEY HOSPITAL

A. McK. FRASER, WILLIAM E. BROWNE, and staff—9. Operative clinic: Gastro-intestinal, biliary tract, thyroid, and hand.

Staff—2. Dry clinic:

WILLIAM E. BROWNE. Results of thyroid surgery on a general surgical service during the past 3 years, accuracy of diagnosis and careful pre-operative preparation, types of operative procedure carried out, end-results.

ROGER C. GRAVES. Management of prostatic obstruction.

WESTON T. BUDDINGTON. Injuries of the ureter.

CHARLES KICKHAM. Non-calculous obstruction of the urethral pelvic junction.

ROBERT H. ALDRICH. English experience with war burns.

JOHN J. TODD. Use of sulfathiazole in surgical infections.

CHARLES A. ROBINSON. Treatment of varicose ulcer.

TIMOTHY E. P. LYONS. Results of 3 years' study of carcinoma of the large bowel.

EDWARD T. WHITNEY. Treatment of fissure in ano.

CHILDREN'S HOSPITAL

WILLIAM E. LADD and associates—9. Dry clinic: Plastic surgery in children, followed by questions and discussion.

WILLIAM E. LADD. Harelip and cleft palate.

DONALD W. MACCOLLUM. Early and late treatment of burns.

WILLIAM E. LADD. Hands, syndactylism, polydactylism.

ROBERT E. GROSS. Plastic operations on the anus.

WILLIAM E. LADD and associates—10:30. Dry clinic, followed by questions and discussions.

THOMAS H. LANMAN. Radical treatment of chronic lung suppuration.

ROBERT E. GROSS. Surgical treatment of patent ductus arteriosus.

FRANC D. INGRAHAM. Subdural hematoma; brain tumors in infancy and childhood.

FAULKNER HOSPITAL

Staff—2. Operative and dry clinic:

E. G. BRACKETT—2. His operation.

H. C. MARBLE—2:15. Wringer hands.

GORDON M. MORRISON—2:30. Bony block in arthritic knees corrected by operation, motion pictures.

OTTO J. HERMANN—2:55. Os calcis fractures, motion pictures.

DR. NEWTON—3:10. Thoracic surgery.

JOHN ADAMS—3:25. Hip cases, drilling and pinning.

H. L. JOHNSON—3:45. Stimulation of fracture repair by embryological regime.

JOHN HOMANS, C. G. MIXTER, R. H. SMITHWICK, DR. WHITE, E. E. O'NEIL, H. C. MARBLE, and J. S. HODGSON—4. Symposium: Causalgia.

COLLIS P. HUNTINGTON MEMORIAL HOSPITAL

Staff—2:15. Dry clinic:

GRANTLEY W. TAYLOR. Choice of treatment in cancer of the oral cavity.

DAVID WEISBERGER. Changes in the oral mucous membrane due to vitamin deficiency.

SHIELDS WARREN. Treatment of leukemia by radioactive substances.

IRA T. NATHANSON. Relation of the hormones to malignant diseases.

MASSACHUSETTS GENERAL HOSPITAL

A. W. ALLEN and associates—8. Symposium: Duodenal ulcer.

C. M. JONES and associates—8. Medical considerations.

A. O. HAMPTON and associates—8:30. Roentgenological diagnosis.

E. B. BENEDICT—8:50. Gastroscopy, gastritis.

A. W. ALLEN—9:10. Surgical considerations.

HOWARD ULFELDER—9:40. Acute perforation.

C. E. WELCH—10:10. Malfunction of anastomotic stoma and after-care.

Staff—10:30. Operative clinic.

V. H. KAZANJIAN—8. Operative and dry clinic: Plastic surgery, presentation of cases operated upon for various deformities.

L. S. MCKITTRICK and associates—2. Operative and dry clinic: Acute small bowel obstruction.

RICHARD WARREN—2. Physiological aspects of non-strangulation obstruction, cause of death.

L. S. MCKITTRICK—2:20. Factors influencing mortality rate.

S. P. SARRIS—2:40. Diagnosis.

RICHARD WARREN—3:10. Non-operative treatment, practical consideration in the use of the Miller-Abbott tube.

L. S. MCKITTRICK—3:30. Résumé, outline of treatment, results.

Staff—3:45. Discussion.

Staff—4. Operations.

MASSACHUSETTS MEMORIAL HOSPITALS

HOWARD M. CLUTE, CLIFFORD D. HARVEY, CHARLES SZIKLAS, FRANK E. BARTON, and HOLLIS L. ALBRIGHT—8. Operative clinic: Thyroid, gall bladder, stomach and colon.

ELEANOR FERGUSON, REGINALD HUNT, and M. JEAN BLACK. Demonstrations: Anesthesia.

Staff—10. Symposium: Sulfonamides.

CHESTER S. KEEFER. Use of sulfonamides in bacteremia.

HOWARD M. CLUTE. Use of sulfonamides in peritoneal cavity.

HOLLIS L. ALBRIGHT. Massive infection of abdominal wall and of the extremities, demonstration of cases.

SAMUEL N. VOSE. Use of sulfonamides in genito-urinary surgery.

LEIGHTON F. JOHNSON. Use of sulfonamides in nose and throat infections.

ARTHUR L. HANRAHAN—10. Operative and dry clinic: Treatment of varicose veins and ulcers.

NEW ENGLAND BAPTIST HOSPITAL

F. H. LAHEY, R. B. CATTELL, S. F. MARSHALL, and B. P. COLCOCK—9. Operative clinic: Thyroid, stomach, colon, and biliary tract.

U. H. EVERSOLE, LEO V. HAND, and M. J. NICHOLSON—9. Anesthesia demonstrations: Regional, spinal, continuous spinal, and general.

H. D. ADAMS and N. W. SWINTON—1:30. Operative clinic: Thyroid, stomach, colon, rectum, and biliary tract.

U. H. EVERSOLE, LEO V. HAND, and M. J. NICHOLSON—1:30. Demonstrations: Anesthesia.

NEW ENGLAND DEACONESS HOSPITAL

F. H. LAHEY, R. B. CATTELL, S. F. MARSHALL, and B. P. COLCOCK—9. Operative clinic: Thyroid, stomach, colon, and biliary tract.

U. H. EVERSOLE, LEO V. HAND, and M. J. NICHOLSON—9. Anesthesia demonstrations: Regional, spinal, continuous spinal, and general.

H. D. ADAMS and N. W. SWINTON—1:30. Operative clinic: Thyroid, stomach, colon, rectum, and biliary tract.

U. H. EVERSOLE, LEO V. HAND, and M. J. NICHOLSON—1:30. Demonstrations: Anesthesia.

F. H. LAHEY, SARA M. JORDAN, and S. F. MARSHALL—2. Dry clinic: Technique of subtotal and total gastrectomy; medical and surgical management of duodenal and gastric ulcer; treatment of gastrojejunal colic fistula.

HERBERT ADAMS. Diagnosis and treatment of regional ileitis.

NEW ENGLAND HOSPITAL FOR WOMEN AND CHILDREN

Staff—10. Out-patient clinics: Gynecological, medical, and pediatric.

G. LINDH MULLER—10. Pathological demonstration: Ovarian tumors.

ELSIE BROWN and ILIA GALLEANI—2. Dry clinic: Metabolism.

NEW ENGLAND MEDICAL CENTER
(Boston Floating Hospital)

Staff—2. Dry clinic:

REEVE H. BETTS and ALFRED WELLER—2. Pulmonary suppuration, treatment of empyema in children.

RICHARD WAGNER and W. S. LEVENSON—2:30. Endocrinology and surgery.

FRANCIS MCDONALD and R. G. FREEMAN—3. Appraisal of the host, nutrition, personality.

FRANCIS MCDONALD, W. S. LEVENSON, NICHOLAS WERTHESSEN, and H. E. MACMAHON—3:30. Case of adrenal dysfunction.

H. E. CHAMBERLIN—4. Genito-urinary surgery in children.

W. A. MACCOLL—4:30. Tonsils and adenoids, indications for removal.

PALMER MEMORIAL HOSPITAL

Staff—9. Symposium: Carcinoma of the rectum and anus.

L. S. MCKITTRICK. Choice of operation in cases of carcinoma of the rectum.

W. F. LEADBETTER. Urological complications after excision of the rectum.

C. C. FRANSEEN. Use of electrocoagulation and radium implantation in poor-risk cases of rectal carcinoma.

J. H. MARKS. X-ray therapy of carcinoma of the rectum.

R. H. SWEET. End-results of treatment of carcinoma of the rectum and anal canal.

ST. ELIZABETH'S HOSPITAL

Staff—9. Operative and dry clinic:

JOSEPH STANTON—9. Cholecystectomy.

JOHN SPELLMAN—9. Resection of large bowel.

JOSEPH STANTON—10. Hysterectomy.

FRANCIS JANTZEN—10. Herniorrhaphy.

WILLIAM SANTORO—11. Cholecystectomy.

MARTIN H. SPELLMAN—11. Herniorrhaphy.

ROGER T. DOYLE—11. Presentation of cases.

JOHN F. CASEY—11:45. The use of sulfonamides in general surgery.

UNITED STATES NAVAL HOSPITAL

JOSEPH B. LOGUE—9. Operations.

LAWRENCE J. MCCARTHY and FRANKLIN G. BALCH, JR.—9. Operative and dry clinic: Gastro-intestinal diseases, partial gastrectomy for pyloric obstruction; synopsis of case histories.

Friday

BETH ISRAEL HOSPITAL

Staff—9. Dry clinic:

C. G. MIXTER. Results of surgery of colon neoplasms classified according to extent of lesion.

L. E. BARRON. Pitfalls in diagnosis of carcinoma of stomach, observations in 300 cases.

C. G. MIXTER. Results of surgery of gastric neoplasms classified according to extent of lesion.

I. T. NATHANSON. Surgical management of lymph nodes in cancer surgery.

KARL PRESSER. Differential diagnosis of gastric ulcer by radiography.

JACOB FINE—10:30. Operation: Colon resection.

C. G. MIXTER—10:30. Operation: Gastric resection.

Staff—2. Dry clinic:

A. M. SELIGMAN. Vitamin K_1 in treatment of hypoprothrombinemia in infants.

J. J. MICHAELS and ETHEL COHEN. Psychiatric considerations in surgical patients.

B. E. F. RISEMAN. The surgical treatment of angina pectoris.

S. A. ROBINS. Diagnosis of non-calcified dermoid cysts.

A. M. SELIGMAN. Carcinogenic agents.

H. L. CABITT. Surgical management of pulmonary tuberculosis.

AARON THURMAN. Thoracic esophageal diverticulum.

NAAMAN STEINBERG. Injection treatment of hemorrhoids.

BOSTON CITY HOSPITAL

Staff—2. Dry clinic:

C. G. SHEDD—2. Chemotherapy in general peritonitis.

MAXWELL FINLAND—2:30. Chemotherapy in surgery.

R. H. ALDRICH—3:15. The dymixal treatment of burns with especial reference to shock.

H. G. DUNPHY—4:15. "Z" plastics in burns.

PETER BENT BRIGHAM HOSPITAL

ELLIOTT C. CUTLER and ROBERT ZOLLINGER—9. Operations: Cholecystectomy, choledochostomy.

ELLIOTT C. CUTLER and associates—10. Symposium: Surgical diseases of the biliary tract.

ELLIOTT C. CUTLER. Biliary calculus, technique of surgical therapy.

MERRILL C. SOSMAN. Upright films of the gall bladder as an aid in the diagnosis of cholelithiasis.

ROBERT ZOLLINGER. Acute cholecystitis.

THOMAS B. QUIGLEY. Surgery of the biliary tract in the aged.

WILLIAM A. DAVIS. Clinical use of vitamin K in surgery of the biliary tract.

EDWARD S. EMERY, JR. Medical treatment after cholecystectomy.

ELLIOTT C. CUTLER and associates—2. Symposium: Surgical treatment of hyperthyroidism.

ELLIOTT C. CUTLER. The domain of thyroid surgery.

SAMUEL A. LEVINE. Pre-operative and postoperative treatment of severe thyrocardiacs.

MARSHALL N. FULTON. Recurrent hyperthyroidism.

J. ENGLEBERT DUNPHY. Use of silk in surgery of the thyroid.

HARRY B. FRIEDGOOD. Rôle of the pituitary hormone in thyrotoxicosis.
ELLIOTT C. CUTLER. Technique of thyroidectomy.

CARNEY HOSPITAL

A. McK. FRASER, WILLIAM E. BROWNE, and staff—9. Operative clinic: Gastro-intestinal, biliary tract, thyroid, and hand.

CHILDREN'S HOSPITAL

WILLIAM E. LADD and associates—9. Operations.

MASSACHUSETTS GENERAL HOSPITAL

E. D. CHURCHILL, OLIVER COPE and associates—9. Symposium: Hyperparathyroidism.
J. C. AUB—9. Physiology of the parathyroid glands.
FULLER ALBRIGHT—9:20. Clinical aspects of hyperparathyroidism.
TRACY B. MALLORY—9:50. Pathology of the parathyroid glands.
OLIVER COPE—10. Surgical treatment of hyperparathyroidism.
F. A. SIMEONE—10:35. Renal complications of hyperparathyroidism.
Staff—11. Operative clinic.
Staff—2. Symposium: Peripheral vascular disease, acute venous thrombosis in veins of lower extremities, pulmonary embolism.
RICHARD WARREN—2. Fundamental considerations of thrombus formation, clinical manifestations, diagnosis and treatment.
H. H. FAXON—2:20. Pulmonary embolism, its relation to venous thrombosis.
RICHARD WARREN—2:45. The use of heparin in the prevention of pulmonary emboli.
C. E. WELCH and C. E. MCGAHEY—3. Venography, indications, technique, results.
H. H. FAXON—3:15. Exploration and division of the femoral vein, indications and operative technique.
C. E. WELCH—3:35. Results, early and late, after division of the femoral vein.
Staff—3:50. Discussion.
H. H. FAXON, C. E. WELCH, and RICHARD WARREN—4. Operative clinic.

MASSACHUSETTS MEMORIAL HOSPITALS

HOWARD M. CLUTE, CLIFFORD D. HARVEY, CHARLES SZIKLAS, FRANK E. BARTON, and HOLLIS L. ALBRIGHT—8. Operative clinic: Thyroid, gall bladder, stomach, and colon.
ELEANOR FERGUSON, REGINALD HUNT, and M. JEAN BLACK. Demonstrations: Anesthesia.
REGINALD HUNT—10:30. Dry clinic: Spinal headaches, preliminary report.
M. JEAN BLACK. Relative efficiency of different methods of oxygen therapy.

NEW ENGLAND BAPTIST HOSPITAL

F. H. LAHEY, R. B. CATTELL, S. F. MARSHALL, and B. P. COLCOCK—9. Operative clinic: Thyroid, stomach, colon, and biliary tract.
U. H. EVERSOLE, LEO V. HAND, and M. J. NICHOLSON—9. Anesthesia demonstrations: Regional, spinal, continuous spinal, and general.
H. D. ADAMS and N. W. SWINTON—1:30. Operative clinic: Thyroid, stomach, colon, rectum, and biliary tract.
U. H. EVERSOLE, LEO V. HAND, and M. J. NICHOLSON—1:30. Demonstrations: Anesthesia.

NEW ENGLAND DEACONESS HOSPITAL

F. H. LAHEY, R. B. CATTELL, S. F. MARSHALL, and B. P. COLCOCK—9. Operative clinic: Thyroid, stomach, colon, and biliary tract.
U. H. EVERSOLE, LEO V. HAND, and M. J. NICHOLSON—9. Anesthesia demonstration: Regional, spinal, continuous spinal and general.
H. D. ADAMS and N. W. SWINTON—1:30. Operative clinic: Thyroid, stomach, colon, rectum, biliary tract.
U. H. EVERSOLE, LEO V. HAND, and M. J. NICHOLSON—1:30. Demonstration: Anesthesia.

NEW ENGLAND HOSPITAL FOR WOMEN AND CHILDREN

Staff—10. Out-patient clinics: Gynecological, ophthalmological, vascular, and pediatric (cardiac).

PALMER MEMORIAL HOSPITAL

Staff—2. Symposium: Diabetic gangrene.
H. F. ROOT. Medical aspects.
L. S. MCKITTRICK. Principles of surgical treatment; classification of lesions; conservative methods.
T. C. PRATT. Indications for and technique of minor amputation.
L. S. MCKITTRICK. Indications for and technique of major amputation.
T. C. PRATT. End-result study of 100 patients after supracondylar amputation for gangrene.
L. S. MCKITTRICK and T. C. PRATT—2. Operations: Supracondylar amputations for gangrene.

ST. ELIZABETH'S HOSPITAL

JOSEPH STANTON and staff—9. Operations.

SALEM HOSPITAL

Staff—10. Clinicopathological conference.

UNITED STATES NAVAL HOSPITAL

CHARLES H. SWAN—9. Operations.

OBSTETRICS AND GYNECOLOGY

Tuesday

BOSTON CITY HOSPITAL

Staff—2. Symposium: Pregnancy, the physiological and pathological aspects.

GEORGE R. MINOT and EUGENE L. LOZNER—2. The hemorrhagic diatheses complicating pregnancy.

FREDERICK J. LYNCH—2:30. Treatment of miscarriage.

FREDERICK PARKER, JR.—3. Pathology of toxemia of pregnancy with special reference to changes in the placenta.

CARMI R. ALDEN—3:30. Disorders of the urinary tract. encountered in obstetrics.

BENEDICT F. BOLAND—4. Hormonal relationship between the kidneys and toxemia of pregnancy.

MAXWELL FINLAND—4:20. Pneumonia in pregnancy.

BOSTON LYING-IN HOSPITAL

Staff—10. Operative and dry clinic:

CHARLES A. JANEWAY. Treatment of puerperal sepsis with sulfanilamide and immune transfusions.

CLEMENT A. SMITH. Quantitative measurements of anesthesia and anoxia produced in mother and infant by obstetrical anesthetics.

CARNEY HOSPITAL

LOUIS E. PHANEUF and staff—9. Operative clinic.

Staff—2. Dry clinic:

LOUIS E. PHANEUF. Manchester operation in management of uterine prolapse.

R. J. HEFFERNAN. Conservative management of premature separation of the placenta.

ROGER C. GRAVES and LOUIS E. PHANEUF. Management of difficult vesicovaginal fistula.

H. EDWARD MACMAHON. Biopsy of uterine cervix from the standpoint of the pathologist.

MAURICE O. BELSON. Uterine hemorrhage; technique of uterovaginal tamponade.

EDMUND L. CAREY. Analysis of breech deliveries at the Carney Hospital during the last 10 years.

WILLIAM C. MALONEY. Abnormal capillary fragility in the newborn.

FREE HOSPITAL FOR WOMEN

JOHN ROCK and SIDNEY C. GRAVES—8. Operations.

Staff—9. Symposium: Carcinoma of the cervix.

FRANK A. PEMBERTON—9. Radium application. Demonstration of radium applicators.

FRANK A. PEMBERTON and RICHARD DRESSER—9:20. Present methods of radium and x-ray treatment.

ARTHUR T. HERTIG and PAUL A. YOUNGE—9:40. Diagnosis of early carcinoma, clinical and pathological.

GEORGE VAN S. SMITH—10:40. Complications and treatment.

PAUL A. YOUNGE—11. Urological complications.

GEORGE VAN S. SMITH—11:20. Results and summary.

PAUL A. YOUNGE—11:45. Hysterectomy for early carcinoma of the cervix.

NEW ENGLAND HOSPITAL FOR WOMEN AND CHILDREN

LETITIA DOUGLAS ADAMS, BLANCHE L. ATWOOD, ILIA GALLEANI, and GRACE E. ROCHFORD—9. Operations.

MARJORIE WOODMAN—2. Obstetrical analgesia.

ESTHER E. BARTLETT—2. Demonstration: Pudendal and parasacral blocks in obstetrical anesthesia.

PALMER MEMORIAL HOSPITAL

G. A. LELAND, JR. and C. J. SWAN—2. Symposium: Treatment of carcinoma of the cervix.

ST. ELIZABETH'S HOSPITAL

Staff—9. Demonstration: Technique of normal delivery.

FREDERICK L. GOOD and staff—10. Operations.

Wednesday

BOSTON CITY HOSPITAL

Staff—9. Dry clinic: Anesthesia and analgesia with special reference to spinal and intravenous anesthesia.

DR. REGINALD and R. D. MARGESON—9:30. Placenta previa in patients previously delivered by low transverse cesarean section.

FREDERICK L. GOOD—10. Physiology of the lower uterine segment.

D. J. MCSWEENEY—10:30. Radiographic pelvimetry.

F. W. O'BRIEN—11. Results in treatment of carcinoma of the uterus in the tumor clinic of a general hospital.

FREDERICK PARKER, JR., and JOHN T. WILLIAMS—11:30. Pathology of so-called fibrosis of the uterus.

BOSTON LYING-IN HOSPITAL

Staff—2. Dry clinic:

MEINOLPH V. KAPPIUS. The rôle of soft tissue x-ray technique in the diagnosis of placenta previa.

ROBERT N. RUTHERFORD. Interpretation of bleeding in the first trimester of pregnancy as shown by endometrial biopsies.

ARTHUR T. HERTIG. Pathological ova, chief cause of spontaneous abortion.

ROBERT H. BARKER. Determination of cephalopelvic disproportion by Thoms method of roentgenometry.

CARNEY HOSPITAL

LOUIS E. PHANEUF and staff—9. Operative clinic.

FREE HOSPITAL FOR WOMEN

EDWARD B. SHEEHAN and CHRISTOPHER J. DUNCAN—8. Gynecological operations.

Staff—9. Dry clinic: Carcinoma of the endometrium.

FRANK A. PEMBERTON—9. Hysterectomy for carcinoma of the endometrium.

FRANK A. PEMBERTON and RICHARD DRESSER—10. Methods of treatment, surgery, radium and x-ray.

ARTHUR T. HERTIG—10:30. Pathology and diagnosis of early carcinoma.

GEORGE VAN S. SMITH—11. Results and summary.

PAUL A. YOUNGE—11:30. Biopsy and cauterization of the cervix in ambulatory patients.

NEW ENGLAND HOSPITAL FOR WOMEN AND CHILDREN

Staff—9. Operations.

MARJORIE WOODMAN—9:30. Obstetrical ward rounds.

MARJORIE WOODMAN—2. Obstetrical analgesia.

ESTHER E. BARTLETT—2. Demonstration: Parasacral and pudendal blocks in obstetrical anesthesia.

PONDVILLE HOSPITAL

JOE V. MEIGS, LANGDON PARSONS, and associates—9. Operative clinic: Wertheim operation for cancer of the cervix; dissection of pelvic lymph nodes (Taussig operation); radium treatment for cancer of the cervix.

ST. ELIZABETH'S HOSPITAL

CHARLES J. KICKHAM—9. Low transverse cervical section; plastic and laparotomy for prolapse of uterus.
FREDERICK L. GOOD and staff—10. Operations.

Thursday

BOSTON LYING-IN HOSPITAL

Staff—10. Operative and dry clinic:
JOHN L. NEWELL. External cephalic version as prophylaxis against the fetal hazards of breech presentations.
DANIEL ABRAMSON. End-results of a conservative policy at delivery as shown in a study of postpartum patients.

CARNEY HOSPITAL

LOUIS E. PHANEUF and staff—9. Operative clinic.

FREE HOSPITAL FOR WOMEN

SIDNEY C. GRAVES and PAUL A. YOUNGE—8. Gynecological operations.
Staff—9. Symposium: Fertility and hormones in gynecology.
JOHN ROCK—9. Technique, interpretation and value of endometrial biopsy.
JOHN O. HAMAN—9:30. Time relationship of ovulation to menstruation.
FRED A. SIMMONS—10. Evaluation and improvement of the male in a sterile mating.
JOHN ROCK—10:30. Clinical lessons from seven previllous embryos.
GEORGE VAN S. SMITH—11. Hormones in gynecology.
W. STEWART WHITTEMORE (from the Cambridge Hospital)—11:30. A case of artificial vagina, pregnancy and cesarean section.
Staff—2. Dry clinic:
PAUL A. YOUNGE—2. Results of presacral neurectomy for pelvic pain.
ROBERT TUCKER—2:30. A follow-up study of cases found to have cholelithiasis at the time of lower abdominal surgery.
PAUL A. YOUNGE—3. The treatment of interstitial cystitis.
FRANK A. PEMBERTON—3:30. The treatment of endometriosis.

MASSACHUSETTS MEMORIAL HOSPITALS

C. WESLEY SEWALL, JOHN J. ELLIOTT, and JOHN C. V. Fisher—8. Operative clinic: Low cervical cesarean, low forceps, mid-forceps; ward walk with demonstration of nursery.
C. WESLEY SEWALL and staff—2. Dry clinic:
JOHN J. ELLIOTT. A ten-year study at the Massachusetts Memorial Hospitals of bleeding in the third trimester of pregnancy.
OWEN C. MULLANEY. Prenatal and postnatal care.
JOHN C. V. FISHER. A ten-year survey of cesarean section at the Massachusetts Memorial Hospitals.
MAX DAVIS. The physiology and clinical application of testosterone.
KENNETH W. SEWALL. A twenty-year survey of eclampsia at the Massachusetts Memorial Hospitals.
HERMAN C. PETTERSON. Modern care of the newborn.

NEW ENGLAND HOSPITAL FOR WOMEN AND CHILDREN

MARJORIE WOODMAN—9:30. Operation: Cesarean. Previous operation on bicornate uterus.
MARJORIE WOODMAN—2. Obstetrical analgesia.
ESTHER E. BARTLETT—2. Demonstration: Pudendal and parasacral blocks in obstetrical anesthesia.

ST. ELIZABETH'S HOSPITAL

Staff—10. Dry clinic and ward visits: Technique of care of breasts in puerperium; review of cesarean sections for the past 10 years.
CHARLES J. KICKHAM—11. Plastic operation for lacerated cervix and perineum.

Friday

CARNEY HOSPITAL

LOUIS E. PHANEUF and staff—9. Operative clinic.

FREE HOSPITAL FOR WOMEN

GEORGE VAN S. SMITH and MEINOLF V. KAPPIUS—8. Gynecological operations.
Staff—9. Operative and dry clinic: Tumors of the ovary.
GEORGE VAN S. SMITH—9. Ovarian tumor.
ARTHUR T. HERTIG—10. Histogenesis and pathology.
RICHARD DRESSER—10:30. X-ray treatment.
FRANK A. PEMBERTON—11. Results in the treatment of carcinoma of the ovary.
SIDNEY C. GRAVES—11:30. Pruritus, kraurosis, leukoplakia and carcinoma of the vulva, etiology, treatment and results.

MASSACHUSETTS GENERAL HOSPITAL

JOE V. MEIGS and associates—9. Operative and dry clinic.
JOE V. MEIGS, LANGDON PARSONS and MARSHALL K. BARTLETT—9. Total abdominal hysterectomy for prolapse; Bassett's operation for cancer of the vulva; modified Kennedy operation for repair of urethral incontinence.
SOMERS STURGIS. Dysmenorrhea, treatment with estrogens.
WARD I. GREGG. Metropathia hemorrhagica, treatment with proluton.
IRA T. NATHANSON. Treatment for painful breasts with testosterone.
LANGDON PARSONS. Carcinoma of the vulva.
MARSHALL K. BARTLETT. Treatment for incontinence.
JOE V. MEIGS. Fibroma of the ovary with fluid in the abdomen and chest.

NEW ENGLAND DEACONESS HOSPITAL

FRANK FOSTER—1:30. Dry clinic: Sterility problem; menstrual abnormalities.
B. P. COLCOCK—1:30. Dry clinic: Endometriosis; severe primary dysmenorrhea.

NEW ENGLAND HOSPITAL FOR WOMEN AND CHILDREN

Staff—9. Operations.
Staff—9:30. Dry clinic: Prenatal and postpartum care.

ST. ELIZABETH'S HOSPITAL

FREDERICK L. GOOD and staff—9. Operations.

UNITED STATES NAVAL HOSPITAL

JOSEPH B. DOYLE—10. Colored motion pictures of spinal anesthesia in obstetrics; discussion of case histories.

Days to be Announced

EVANGELINE BOOTH MATERNITY HOSPITAL

A. K. PAINE, W. J. MCDONALD, H. S. FINKEL, M. G. BERLIN, J. E. HOPKINS, D. C. GOLDFARB, A. A. LEVI, L. ALBERT, S. ELLIS, J. TARTAKOFF, and S. SIDELL. Operative and Dry Clinics.

THORACIC SURGERY

Monday

MASSACHUSETTS MEMORIAL HOSPITALS

JOHN W. STRIEDER and W. W. WOODRUFF (Saranac Lake, New York)—2. Symposium: Thoracic surgery. Suppurative pericarditis, colored moving pictures; lobectomy for bronchiectasis; tuberculous empyema.

NEW ENGLAND BAPTIST HOSPITAL

H. D. ADAMS—1:30. Operations.

NEW ENGLAND DEACONESS HOSPITAL

RICHARD H. OVERHOLT and REEVE H. BETTS.—2. Operative clinic: Non-tuberculous thoracic surgery.

Tuesday

NEW ENGLAND BAPTIST HOSPITAL

H. D. ADAMS—1:30. Operations.

NEW ENGLAND DEACONESS HOSPITAL

Staff—2. Dry clinic: Symposium on total pneumonectomy.
REEVE H. BETTS. Determination of operability in primary carcinoma of the lung.
SHIELDS WARREN. Pathology of lung cancer.
RICHARD H. OVERHOLT. Exploratory thoracotomy in suspected malignancy.
BERT H. COTTON. Use of sulfathiazole in pulmonary resection.
ANDREW YEOMANS. Physiological observations made at time of pulmonary resection.
PHILLIP SCHULTZ. Problems of anesthesia in pneumonectomy.
HOWARD ROOT. Medical aspects of postoperative management.
Staff. Presentation of cases treated by pneumonectomy for cancer, tuberculosis, bronchiectasis, and cystic disease.

Wednesday

COREY HILL HOSPITAL

Staff—2. Operative clinic: Pulmonary tuberculosis.

MASSACHUSETTS GENERAL HOSPITAL

E. D. CHURCHILL and associates—9. Symposium and clinic: Primary tumors of the lung and esophagus.
D. S. KING—9. Medical aspects of primary carcinoma of the lung.
E. D. CHURCHILL—9:20. Types of operations and results in carcinoma of the lung.
R. H. SWEET—9:50. Resection of the lung for metastatic sarcoma.
R. H. SWEET—10. Surgical treatment of carcinoma of the lower third of the esophagus and cardiac area of the stomach.
E. D. CHURCHILL—10:30. Surgical treatment of carcinoma of the upper two-thirds of the esophagus.
ROBERT KLOPSTOCK—10:45. Discussion and exhibition of gross pathological specimens.
Staff—11. Operations.

MASSACHUSETTS MEMORIAL HOSPITALS

JOHN W. STRIEDER—2. Operative clinic: Lobectomy for bronchiectasis; thoracoplasty for tuberculosis.

NEW ENGLAND BAPTIST HOSPITAL

H. D. ADAMS—1:30. Operations.

NEW ENGLAND DEACONESS HOSPITAL

H. D. ADAMS—1:30. Operations.

Thursday

MASSACHUSETTS GENERAL HOSPITAL

E. D. CHURCHILL and associates—2. Operative and dry clinic: Suppurative lesions of the lung.
D. S. KING—2. Medical aspects of bronchiectasis, prognosis of untreated cases.
L. DAVENPORT—2:20. Technique of bronchography and anatomy of bronchial tree.
E. D. CHURCHILL—2:40. The surgical treatment of bronchiectasis.
R. H. SWEET—3. Surgical treatment of lung abscess, results.
E. D. CHURCHILL—3:30. Lobectomy and pneumonectomy in lung abscess.
ROBERT KLOPSTOCK—3:45. Discussion and exhibition of gross pathological specimens.

NEW ENGLAND BAPTIST HOSPITAL

H. D. ADAMS—1:30. Operations.

Friday

BOSTON CITY HOSPITAL

Staff—9. Operative and dry clinic:
J. W. STRIEDER—9. Colored motion pictures of suppurative pericarditis; result of modern method of treatment of putrid empyema; lobectomy for bronchiectasis.
HORACE BINNEY—9:45. Methods and results of treatment of pulmonary tuberculosis.
S. J. G NOWAK—10:45. Experimental pulmonary embolus.
DR. SHULTZ—11:30. Intratracheal anesthesia for thoracic surgery.

NEW ENGLAND DEACONESS HOSPITAL

Staff—2. Symposium on total pneumonectomy.
REEVE H. BETTS. Determination of operability in primary carcinoma of the lung.
SHIELDS WARREN. Pathology of lung cancer.
RICHARD H. OVERHOLT. Exploratory thoracotomy in suspected malignancy.
BERT H. COTTON. Use of sulfathiazole in pulmonary resection.
ANDREW YEOMANS. Physiological observations made at time of pulmonary resection.
PHILLIP SCHULTZ. Problems of anesthesia in pneumonectomy.
HOWARD ROOT. Medical aspects of postoperative management.
Staff. Presentation of cases treated by pneumonectomy for cancer, tuberculosis, bronchiectasis, and cystic disease.

NEW ENGLAND BAPTIST HOSPITAL

H. D. ADAMS—1:30. Operations.

ORTHOPEDIC SURGERY

Tuesday

CHILDREN'S HOSPITAL

FRANK R. OBER and associates—9. Operations.

NEW ENGLAND BAPTIST HOSPITAL

G. E. HAGGART and J. W. TOUMEY, JR.—9. Operations.

NEW ENGLAND DEACONESS HOSPITAL

G. E. HAGGART and J. W. TOUMEY, JR.—9. Operations.

Wednesday

ROBERT BRECK BRIGHAM HOSPITAL

Staff—9. Operative Clinic:
JOHN G. KUHNS. Arthroplasty of elbow.
ROBERT J. JOPLIN. Posterior capsularplasty of knee for flexion deformity.
WILLIAM A. ELLISTON. Osteotomy for malum coxae senilis.
Staff—2. Symposium: End-results in correction of arthritic deformities.
LORING T. SWAIM. Treatment of spinal arthritis.
JOHN G. KUHNS. Correction of contractures of the knee.
ROBERT J. JOPLIN. End-results in arthroplasties of elbow.
JOHN A. REIDY. Arthroplasties of knee.
WILLIAM A. ELLISTON. Treatment of osteo-arthritis of hip.

CARNEY HOSPITAL

W. R. MACAUSLAND and staff—9. Operative clinic.

CHILDREN'S HOSPITAL

Staff—9. Dry clinic:
WILLIAM A. ELLISTON. Study in bone growth and resorption of the epiphyseal plate.
LEO J. MCDERMOTT. Equalization of leg length; leg lengthening.
PAUL W. HUGENBERGER. Tuberculosis of bone.
ALBERT H. BREWSTER. Arthrodesis of the foot in spastic paralysis.
Staff—11. Symposium: Infantile paralysis, operative treatment, round-table discussion.

MASSACHUSETTS MEMORIAL HOSPITALS

LOUIS G. HOWARD, KENNETH CHRISTOPHE, and WILLIAM A. ELLISTON—9. Operative clinic: Spinal fusion; hip reconstruction with vitallium cup.

NEW ENGLAND BAPTIST HOSPITAL

G. E. HAGGART and J. W. TOUMEY, JR.—9. Operations.

NEW ENGLAND DEACONESS HOSPITAL

G. E. HAGGART and J. W. TOUMEY, JR.—9. Operations.

NEW ENGLAND HOSPITAL FOR WOMEN AND CHILDREN

MIRIAM G. KATZEFF—9. Operations. Dry clinic: Arthritis, fractures, x-rays of pathological bone conditions, clubfoot demonstration.

ST. ELIZABETH'S HOSPITAL

THOMAS F. BRODERICK—9. Spinal fusion. Dry clinic: Motion pictures of author's technique of spinal fusion.

UNITED STATES NAVAL HOSPITAL

THOMAS H. PETERSON—9. Dry clinic: Shoulder injuries; discussion of case histories; x-ray findings.

Thursday

CHILDREN'S HOSPITAL

FRANK R. OBER and associates—9. Operations.

FAULKNER HOSPITAL

E. G. BRACKETT, H. C. MARBLE, G. M. MORRISON, J. L. DOHERTY, J. D. ADAMS, and OTTO J. HERMANN—8. Operative clinic.
HARVEY MORRISON. X-ray demonstrations.

MASSACHUSETTS GENERAL HOSPITAL

M. N. SMITH-PETERSEN and associates—2. Operative and dry clinic:
M. N. SMITH-PETERSEN, ARMIN KLEIN, J. S. BARR, and PAUL L. NORTON—2. Operations.
WILLIAM A. ROGERS—2. Fractures and fracture dislocations of the cervical spine, results of operative treatment.
GEORGE VAN GORDER. Amputations.
E. F. CAVE. Fractures of the carpal scaphoid, results of bone graft.
O. SHERWIN STAPLES. Effect on the knee joint of panastragalar arthrodesis.
OTTO E. AUFRANC, CARROLL B. LARSON, and M. N. SMITH-PETERSEN. Surgical procedures for relief of deformities arising from rheumatoid arthritis.
WALTER BAUER. Discussion.

MASSACHUSETTS MEMORIAL HOSPITALS

LOUIS G. HOWARD, ALBERT B. FERGUSON, and THOMAS J. ANGLEM—9. Discussion of malignant bone tumors.

NEW ENGLAND BAPTIST HOSPITAL

G. E. HAGGART and J. W. TOUMEY, JR.—9. Operations.

NEW ENGLAND DEACONESS HOSPITAL

G. E. HAGGART and J. W. TOUMEY, JR.—9. Operations.

ST. ELIZABETH'S HOSPITAL

THOMAS F. BRODERICK and staff—9. Operations.

Friday

CARNEY HOSPITAL

W. R. MACAUSLAND and staff—9. Operative clinic.

CHILDREN'S HOSPITAL

Staff—9. Dry clinic followed by round-table discussion:
MIRIAM G. KATZEFF—9. Early treatment of clubfeet.
ROBERT H. MORRIS—9:20. Treatment of difficult clubfeet.
FRANK R. OBER—9:50. Treatment of congenital elevation of the scapula.
WILLIAM T. GREEN—10:20. Osteochondritis dissecans.
MEIER G. KARP—10:45. Fractures about the elbow.
Staff—11:15. Congenital malformations.

MASSACHUSETTS GENERAL HOSPITAL

M. N. SMITH-PETERSEN and associates—8:30. Operative and dry clinic:

G. W. VANGORDER, W. A. ROGERS, and E. F. CAVE—8:30. Operations.
ARMIN KLEIN, JOHN A. REIDY, and ROBERT J. JOPLIN—9. Slipped upper femoral epiphysis, results of operative and non-operative treatment.
JOSEPH S. BARR. Spinal fusion, frequency of pseudarthrosis.
M. N. SMITH-PETERSEN, CARROLL E. LARSON, and OTTO E. AUFRANC. Arthroplasty of the hip, results.

NEW ENGLAND BAPTIST HOSPITAL

G. E. HAGGART and J. W. TOUMEY, JR.—9. Operations.

NEW ENGLAND DEACONESS HOSPITAL

G. E. HAGGART and J. W. TOUMEY, JR.—9. Operations.

UNITED STATES NAVAL HOSPITAL

THOMAS H. PETERSON—9. Operations.

GENITO-URINARY SURGERY

Tuesday

BETH ISRAEL HOSPITAL

Staff—9. Dry clinic:
E. G. CRABTREE. Pre-operative preparation of prostatic cases.
G. C. PRATHER and M. L. BRODNY. Accessory bladder pathology in prostatic cases.
G. C. PRATHER and MORTIMER REICH. Accessory renal pathology in prostatic cases.
E. G. CRABTREE. Summary of 12 years' experience in prostatic surgery.
G. C. PRATHER. Results of contact therapy for bladder tumors.
E. G. CRABTREE—10:45. Operation: Bladder tumor, contact radiation therapy.
Staff—2. Dry clinic:
E. L. PRIEN. Adaptation of the Tratner hydrophorograph for bladder studies.
ABRAHAM ZIMMERMAN. Bladder atonies.
J. H. LIPTON. Bladder atonies as demonstrated by the Tratner hydrophorograph.
S. R. MUELLNER. Testosterone effect on bladder tone; diabetic atony of the bladder.
B. E. GREENBERG. Value of cholinergic and adrenergic drugs in bladder atonies.
H. A. KONTOFF. Rate of recovery of bladder after relief of obstruction.
S. R. MUELLNER. Stress incontinence in the female.
E. L. PRIEN. Crystallography of the sulfonamide drugs.

CHILDREN'S HOSPITAL

WILLIAM E. LADD and associates—9. Dry clinic: Diseases of the genito-urinary tract in infancy and childhood, followed by questions and discussion.
WILLIAM E. LADD. Embryoma of the kidney (Wilms' tumor).
ROBERT E. GROSS. Surgical treatment of malformations.
THOMAS H. LANMAN. Exstrophy and epispadias.

MASSACHUSETTS MEMORIAL HOSPITALS

Staff—9. Operative clinic:
M. J. HAHN—9. Pyelotomy for stone.
DAVID B. STEARNS—10. Suprapubic prostatectomy.
SAMUEL N. VOSE—11. Perineal prostatectomy.

NEW ENGLAND BAPTIST HOSPITAL

E. E. EWERT and VERNON S. DICK—9. Operations.

NEW ENGLAND DEACONESS HOSPITAL

E. E. EWERT and VERNON S. DICK—9. Operations.

ST. ELIZABETH'S HOSPITAL

EDWARD J. O'BRIEN and associates—10. Operations.

Wednesday

MASSACHUSETTS GENERAL HOSPITAL

G. G. SMITH and associates—9. Operative and dry clinic:
Staff—9. Operations.
F. H. COLBY—10:30. Genito-urinary tuberculosis.
RICHARD CHUTE—10:50. Results of treatment with estrogenic substances in prostatic hypertrophy.
S. B. KELLEY—11:10. Complications of transurethral resection.
LORANDE WOODRUFF—11:40. The female urethra.
G. G. SMITH and associates—2. Operative and dry clinic;
Staff—2. Operations.
G. G. SMITH—3:30. Treatment of prostatic cancer.
ARTHUR WILLETS—3:50. Chronic prostatitis.
WYLAND LEADBETTER—4:10. Renal arteriography.
JOHN GENS—4:30. Urological endocrinology.

MASSACHUSETTS MEMORIAL HOSPITALS

Staff—9. Symposium: Obstructing prostate.
CLYDE L. DEMING (New Haven, Connecticut). Functional end-results following perineal prostatectomy.
EDWARD N. COOK (Rochester, Minnesota). Pre-operative preparation for transurethral prostatectomy.
SAMUEL N. VOSE. Selection of operative procedure.
JOSEPH F. MCCARTHY (New York). Technical aspects of prostatic resection with commentary on pharmacological adjuvants.

NEW ENGLAND BAPTIST HOSPITAL

E. E. EWERT and VERNON S. DICK—9. Operations.

NEW ENGLAND DEACONESS HOSPITAL

HARVARD H. CRABTREE—9. Operations.
E. E. EWERT and VERNON S. DICK—9. Operations.

NEW ENGLAND HOSPITAL FOR WOMEN AND CHILDREN

SUSANNAH FRIEDMAN—2. Dry clinic: End-results of pyelitis of pregnancy.

NEWTON HOSPITAL

E. G. CRABTREE, G. C. PRATHER, and E. L. PRIEN—9. Operations.
Staff—2. Dry clinic:
E. L. PRIEN. Analysis of calcifications and crystals in renal tissue.
G. C. PRATHER. Cystein stone and cysteinuria.
E. G. CRABTREE. Block dissections for renal tumor and cancer of the bladder.
G. C. PRATHER. Calyceal diverticula.
E. L. PRIEN. Multiple recurrent stones, palliation.
E. G. CRABTREE. Uretero-nephrectomy.
G. C. PRATHER. Graphic evidence of upper urinary dilatation with lower urinary obstruction.

PONDVILLE HOSPITAL

ROGER C. GRAVES and associates—9. Operative clinic: Carcinoma of the bladder.
Staff—11. Dry clinic:
ROGER C. GRAVES. Carcinoma of the bladder.
CHARLES C. J. KICKHAM. Urological complications in carcinoma of the rectum and colon.
WESTON T. BUDDINGTON. Urological complications in carcinoma of the cervix.

Thursday

BETH ISRAEL HOSPITAL

Staff—9. Dry clinic:
E. L. PRIEN. A new method for stone analysis.
D. B. STEARNS. Parathyroid disease and urinary lithiasis.
G. C. PRATHER. Roentgenology of kidney at operation.
B. E. GREENBERG. Use of cholinergic and adrenergic medication for ureteral stone passage.
H. A. KONTOFF. Hospital statistics on urinary lithiasis.
J. H. LIPTON. Clearance of infection in stone cases after operation.
D. B. STEARNS. Diagnosis of calyceal calcifications.
H. A. KONTOFF. Renal tumor statistics of the hospital. Demonstration of surgical specimens of renal tumors with pyelographic and microscopic data.
E. G. CRABTREE—11. Operation: Excision of renal tumor.
Staff—2. Dry clinic:
M. L. BRODNY. Fractional phylography.
J. H. LIPTON. Sulfonamide drugs in surgical wounds and postoperative infections.
B. E. GREENBERG. Methylene blue treatment for tuberculosis of the bladder.
E. L. PRIEN. Renal injury from sulfathiazole and sulfapyridine, nature of the lesion.
D. B. STEARNS. Undescended testicle, age groups.
M. L. BRODNY. Urological study of enuresis cases.
DR. FISHMAN. Denervation of the kidney, end-results of 200 cases.
B. E. GREENBERG. Organization and function of a sterility clinic.
NATHAN CHASET. Hypertensive disease in urology.

BOSTON CITY HOSPITAL

Staff—9. Operative and dry clinic:
H. H. HOWARD—9. Epidermoid carcinoma of the penis.
GEORGE C. PRATHER—9:25. Stricture of the urethra.
S. R. MUELLNER—9:50. Newer concepts of urinary extravasation.
G. D. ATKINSON—10:15. Scrotal plastics after idiopathic gangrene.
W. H. HOLTHAM—10:40. Treatment of gonorrheal urethritis with sulfathiazole.
J. A. SETH—11:05. Removal of calculi from lower ureter by the vaginal route, report of 2 cases.
F. G. SHEDDAN—11:25. Fracture of the penis, case report. Anatomical study of remnants of prostate tissue in the prostatic bed following enucleation.
AUGUSTUS RILEY—11:40. Hemangioma of kidney.

PETER BENT BRIGHAM HOSPITAL

WILLIAM C. QUINBY and associates—2. Symposium:
J. HARTWELL HARRISON. The diagnosis and treatment of common injuries of the urinary tract.
GEORGE AUSTEN. A correlation of the clinical aspects and pathology of the fused renal mass.
G. PHILIP GRABFIELD. Renal blood flow.
F. ANTHONY SIMEONE. Experimental hypertension.
WILLIAM C. QUINBY. A critical evaluation of prostatic surgery in recent years.

CARNEY HOSPITAL

ROGER C. GRAVES and staff—9. Operative clinic.

MASSACHUSETTS GENERAL HOSPITAL

G. G. SMITH and associates—9. Operative and dry clinic:
Staff—9. Operations.
HOWARD SUBY—10:30. Solution of urinary calculi.
RICHARD CHUTE—10:50. Problems in the treatment of urinary calculi.
F. H. COLBY—11:15. Deep x-ray therapy in cancer of the bladder.
S. B. KELLEY—11:40. Ureterostomy versus nephrostomy.

NEW ENGLAND BAPTIST HOSPITAL

E. E. EWERT and VERNON S. DICK—9. Operations.

NEW ENGLAND DEACONESS HOSPITAL

E. E. EWERT and VERNON S. DICK—9. Operations.

UNITED STATES NAVAL HOSPITAL

NICHOLAS G. SCARCELLO—9. Dry clinic: Presentation of urological cases with histories and x-ray findings.

Friday

MASSACHUSETTS GENERAL HOSPITAL

R. H. SMITHWICK and associates—2. Symposium: Surgery of non-nephritic hypertension.
ROBERT S. PALMER. Statement of the problem.
BENJAMIN CASTLEMAN. Renal pathology.
JOHN S. TALBOT. Renal physiology.
RICHARD CHUTE. Urological factors.
R. H. SMITHWICK. Modification of abnormal physiology by sympathectomy.

MASSACHUSETTS MEMORIAL HOSPITALS

Staff—9. Dry clinic:
DAVID B. STEARNS. Prostatovesiculitis simulating upper urinary tract symptomatology.
SAMUEL N. VOSE. Radical prostatectomy for cancer of prostate.
FRANK MORSE, JR. Congenital bladder neck obstruction in male children.
M. F. HAHN, JR. Review of urinary calculus cases at the Massachusetts Memorial Hospitals during past 15 years.

NEW ENGLAND BAPTIST HOSPITAL

E. E. EWERT and VERNON S. DICK—9. Operations.

NEW ENGLAND DEACONESS HOSPITAL

E. E. EWERT and VERNON S. DICK—9. Operations.

NEW ENGLAND HOSPITAL FOR WOMEN AND CHILDREN

G. LINDH MULLER—10. Pathological demonstration: Congenital abnormalities of the kidney, nephrolithiasis.

FRACTURES AND OTHER TRAUMAS

Tuesday

BOSTON CITY HOSPITAL

OTTO J. HERMANN and associates—9. Symposium: Fractures of the ankle.
OTTO J. HERMANN. Introduction.
GORDON M. MORRISON. Simple unilateral ankle injuries with fracture.
JOSEPH BURNETT, G. G. BAILEY, JOSEPH SHORTELL, and RUSSELL SULLIVAN. Complicated ankle fractures, bilateral, following Cotton's therapeutical ankle classifications. Slides, charts, individual cases, and apparatus illustrating various points.

Thursday

BEVERLY HOSPITAL

Staff—9. Operative and dry clinic:
PEER P. JOHNSON. Interesting fractures, end-results.
JOHN D. ADAMS. Injuries to hip joint; drilling of femoral neck in dislocation of hip joint.
RICHARD E. ALT. Open reduction in fractures and dislocations.
PAUL E. TIVNAN and JOSEPH A. CUNNINGHAM. Clinico-pathological conference on bone tumors.
JACOB H. FINE. Demonstration of recently constructed operating amphitheater eliminating fire and explosion hazards.

Friday

BOSTON CITY HOSPITAL

Staff—9. Symposium: Fractures of the hip.
NEWTON C. BROWDER—9. Intracapsular fractures of the hip.
RUSSELL SULLIVAN—9:30. Hip fractures, cases of operative reduction and internal fixation.
OTTO J. HERMANN—10. Painful ununited hip fractures.
THOMAS H. PETERSON—10:30. Posterior fusion of the hip.
A. A. THIBODEAU—11. Slipped femoral epiphysis.
JAMES W. SEVER—11:30. Presentation of cases.
DR. WISE (of New York)—11:45. Presentation of autopsy specimens of nailed fractured hips.

MASSACHUSETTS GENERAL HOSPITAL

A. W. REGGIO, A. W. ALLEN, M. N. SMITH-PETERSEN, H. C. MARBLE, G. W. VANGORDER, and associates—2. Dry clinic.
Staff—2. Fracture rounds; presentation of current cases with open discussion.
Staff—3:30. End-result clinic; presentation of follow-up cases with open discussion of problems.

UNITED STATES MARINE HOSPITAL

RICHEY L. WAUGH and associates—8. Operative clinic: Internal derangement of knee-joint; bunion, closed treatment, double-pin method; fracture both bones of leg, open treatment using vitallium plate.
RICHEY L. WAUGH and associates—1. Dry clinic:
RICHEY L. WAUGH. Skeletal traction and countertraction methods applicable to ordinary extension splints in the treatment of fractures of lower extremity.
THOMAS A. HATHCOCK. Use of vitallium in fractures.
RICHEY L. WAUGH. Injuries of the wrist.

NEUROSURGERY

Monday

MASSACHUSETTS GENERAL HOSPITAL

J. C. WHITE and associates—2. Symposium: Neurological surgery.
J. C. WHITE and J. J. MICHELSEN—2. Operations.
W. J. MIXTER—4. Electro-encephalographic control of anticonvulsant therapy.
C. S. KUBIK and A. O. HAMPTON—4. Removal of lipiodol by lumbar puncture.
J. C. WHITE—4. Effect of anesthesia on volume of brain.

Tuesday

MASSACHUSETTS GENERAL HOSPITAL

J. C. WHITE and associates—9. Operative and dry clinic:
W. J. MIXTER and J. S. HODGSON—9. Operations.
J. C. WHITE—11. Presentation of operative results in angina pectoris and extrapyramidal tremors.
J. J. MICHELSEN—11. Treatment of torticollis by alcohol injection of cervical muscles.
J. C. WHITE and associates—2. Dry clinic:
W. J. MIXTER. Protrusion of nucleus pulposus in cervical spine.
J. S. HODGSON. Experience with cerebellar hemangiomas (Lindau's disease).
J. J. MICHELSEN. Subdural abscess.
W. H. SWEET. New methods for measurement of blood flow.
J. C. WHITE. Spinothalamic tractotomy in the medulla.

Wednesday

BOSTON CITY HOSPITAL

DONALD MUNRO and associates—9. Symposium: Injuries of the central nervous system and allied conditions.
DONALD MUNRO—9. Treatment of compound fracture of the skull, a study of 175 cases.
WALTER WEGNER—9:20. Concomitant cerebral tumors and craniocerebral injuries.
TIMOTHY LEARY—9:40. Pathology of craniocerebral injuries, with demonstration of specimens.
DONALD MUNRO—10:40. Study of 300 consecutive intracranial subdural hematomas.
WALTER WEGNER—11. Care of bone damage in cervical spinal cord injuries.
DONALD MUNRO—11:15. Present-day tidal drainage of the urinary bladder.
C. W. ELKINS—11:30. Two-needle oxygen spinograms with improved visualization of the spinal subarachnoid space.
DONALD MUNRO—11:45. Study of end-results in 130 cases of all levels of spinal cord injury.

DONALD MUNRO—12:10. Spinothalamic tractotomy in the medulla, intradural differential section of the sensory root of the fifth cranial nerve and other procedures of use in dealing with intractable pain in the head, face, mouth, neck, and arms.
DONALD MUNRO—12:20. Differential diagnosis and treatment of Ménière's disease and aural vertigo.
DONALD MUNRO—12:30. Prophylaxis and treatment of pressure bed-sores.

Thursday

CHILDREN'S HOSPITAL

F. D. INGRAHAM and associates—9. Symposium: Neurological surgery of children.
F. D. INGRAHAM—9. Neurosurgical problems of infancy and childhood.
BRONSON CROTHERS—9:30. Definition of recovery in terms of growth and development.
F. D. INGRAHAM—9:45. Congenital anomalies requiring surgical treatment.
F. D. INGRAHAM—10:15. Technical aids in neurosurgery of infancy and childhood.
BRONSON CROTHERS—10:30. Reliability of encephalography in infancy.
F. D. INGRAHAM—10:45. Presentation of cases.
SYDNEY FARBER—11. Pathological aspects.
F. D. INGRAHAM—11:30. Excision of premotor cortex for athetosis.
F. D. INGRAHAM—12. Subdural hematoma.

NEW ENGLAND DEACONESS HOSPITAL

GILBERT HORRAX and associates—2. Operations:
GILBERT HORRAX. Ventriculogram and craniotomy for brain tumor.
J. L. POPPEN. Herniated intervertebral disc.

Friday

NEW ENGLAND DEACONESS HOSPITAL

GILBERT HORRAX and associates—9. Dry clinic:
J. L. POPPEN—9. Results in herniated intervertebral discs, operations with and without fusions.
J. L. POPPEN—9:30. Platybasia demonstration.
GILBERT HORRAX—10. Experiences and results with brain tumors for the last 8 years.

OTORHINOLARYNGOLOGY

Monday

NEW ENGLAND BAPTIST HOSPITAL

F. D. LATHROP—1:30. Operations.

NEW ENGLAND DEACONESS HOSPITAL

W. B. HOOVER and F. D. LATHROP—2:30. Local anesthesia in tonsillectomy, adenoidectomy, lingual tonsillectomy, and excision of lateral pharyngeal bands; septum; sinus surgery—Caldwell-Luc, external frontal, intranasal; plastic procedures, external nose.

NEW ENGLAND MEDICAL CENTER

(Joseph H. Pratt Diagnostic Clinic—Boston Dispensary)
GEORGE KELEMEN—2. Pathogenesis of traumatic cholesteatome, lantern slides; surgery of the tonsil after middle-life; pathways of infection in the ear, lantern slides.
PHILIP E. MELTZER—2. Surgical anatomy of the temporal bone.

Tuesday

BOSTON CITY HOSPITAL

Staff—9. Dry clinic:
E. J. MONAHAN—9:30. A method of the extirpation of bronchial cysts.
OSCAR HIRSCH—9:50. Cysts of pituitary body.
LOUIS M. FREEDMAN—10:10. Bronchoscopic treatment of pulmonary atelectasis.
CHESTER R. MILLS—10:30. Osteomyelitis of the frontal bones.
BENJAMIN RISEMAN—10:50. Gumma of nasopharynx and lung.
SAMUEL W. GARFIN—11:10. Treatment of fractures of the jaw.
PHILIP E. A. SHERIDAN, CHARLES DEWOLFE and FREDERICK HEIMLICK—11:30. Presentation of cases.

CHILDREN'S HOSPITAL

Staff—9:30. Dry clinic:
CHARLES F. FERGUSON. Acute laryngotracheal bronchitis, report and statistical study of 40 cases.
C. G. FLAKE. Congenital anomalies of the tracheal bronchial tract; treatment of choanal atresia.
C. G. FLAKE and CHARLES F. FERGUSON. Chemotherapy in upper respiratory infections of children.

MASSACHUSETTS EYE AND EAR INFIRMARY

Staff—2:30. Dry clinic:
A. S. MACMILLAN. Interpretation of sinus x-rays.
L. G. RICHARDS. Problems in bronchoscopy.
C. T. PORTER. Orbital abscess, diagnosis and treatment.
L. A. SCHALL. Cancer of the nose and sinuses.

MASSACHUSETTS MEMORIAL HOSPITALS

Staff—2. Operative clinic:
HAROLD L. BABCOCK. Mastoidectomy.
BARNET WEIN. Radical sinus operation.
LEIGHTON F. JOHNSON. Fenestration for otosclerosis.

NEW ENGLAND BAPTIST HOSPITAL

WALTER B. HOOVER—9. Operations.
F. D. LATHROP—1:30. Operations.

NEW ENGLAND DEACONESS HOSPITAL

WALTER B. HOOVER—9. Operations.
F. D. LATHROP—1:30. Operations.

NEW ENGLAND HOSPITAL FOR WOMEN AND CHILDREN

ELIZABETH DEBLOIS, ISABELLE D. KERR, MARGARET NOYES KLEINERT, and MARY F. VASTINE—10. Demonstration: Use of the audiometer.

ST. ELIZABETH'S HOSPITAL

JOHN E. BURNS—9. Operative clinic: Radical frontal sinus; radical mastoidectomy.

UNITED STATES NAVAL HOSPITAL

CHARLES H. ALLMAN—9. Dry clinic: Bronchoscopy for bronchiectasis; pulmonary abscess and postoperative atelectasis; synopsis of case histories, end-results.
DAVID P. CORDRAY—9. Operations.

Wednesday

MASSACHUSETTS EYE AND EAR INFIRMARY

Staff—2:30. Dry clinic:
G. B. FRED. Osteomyelitis of the frontal bone.
H. G. TOBEY. Allergy, otolaryngological considerations.
P. E. MELTZER and C. I. JOHNSON. The fenestration operation.
RUTH GUILDER. The deaf child.
A. S. MACMILLAN. Interpretation of mastoid x-rays.

MASSACHUSETTS MEMORIAL HOSPITALS

LEIGHTON F. JOHNSON—2. Dry clinic: Mediastinitis, 3 cases, 2 cures (with Howard M. Clute); endaural repair of postauricular fistula, 3 cases; osteomyelitis of frontal bone; Lempert's operation, endaural fenestration for otosclerosis; ears in contagious diseases (with Conrad Wesselhoeft).

NEW ENGLAND BAPTIST HOSPITAL

WALTER B. HOOVER—9. Operations.
F. D. LATHROP—1:30. Operations.

NEW ENGLAND DEACONESS HOSPITAL

WALTER B. HOOVER—9. Operations.
WALTER B. HOOVER and F. D. LATHROP—2:30. Dry clinic: Diseases of the esophagus, summary of symptoms, diagnosis and treatment; carcinoma; cardiospasm; esophageal diverticulum; esophagitis; ulcers, strictures and perforation of the esophagus.

Thursday

BETH ISRAEL HOSPITAL

Staff—9. Dry clinic:
SAMUEL CLINE. Tuberculous laryngitis.
OSCAR HIRSCH. Sphenoid approach through submucous septum.
A. J. GORNEY. Lempert's simple and radical mastoid operations.
S. W. GARFIN. Transseptal approach for dacryocystitis.
H. F. FRIEDMAN. Radiation for carcinoma of larynx by the Coutard method.
BENJAMIN RISEMAN. Cavernous sinus thrombosis.
LOUIS M. FREEDMAN. Coutard radiation therapy for carcinoma of larynx, end-results; Lempert technique for simple and radical mastoidectomy.
LOUIS M. FREEDMAN. Lempert operation for mastoiditis.

BOSTON CITY HOSPITAL

Staff—9. Operations.

CARNEY HOSPITAL

BENJAMIN E. RISEMAN—2. Edema of larynx with abscess.
CORNELIUS S. HICKEY—2. Mastoiditis with complications.

MASSACHUSETTS EYE AND EAR INFIRMARY

Staff—9. Operations.
Staff—2:30. Dry clinic:
L. A. SCHALL. Laryngeal cancer.
V. H. KAZANJIAN. Rhinoplastic surgery.
PHILIP MYSEL. Labyrinthectomy, review of cases.
C. M. KOS. Meningitis, review of cases and discussion.

NEW ENGLAND BAPTIST HOSPITAL

WALTER B. HOOVER—9. Operations.
F. D. LATHROP—1:30. Operations.

NEW ENGLAND DEACONESS HOSPITAL

WALTER B. HOOVER—9. Operations.

NEW ENGLAND HOSPITAL FOR WOMEN AND CHILDREN

ELIZABETH DEBLOIS, ISABELLE D. KERR, MARGARET NOYES KLEINERT, and MARION C. SABIA—9. Operations.
Staff—10. Demonstration: Use of the audiometer.

ST. ELIZABETH'S HOSPITAL

WILLIAM T. HALEY—9. Operations.

Friday

BETH ISRAEL HOSPITAL

Staff—9. Operations.

CARNEY HOSPITAL

BENJAMIN E. RISEMAN and staff—9. Operative clinic.

MASSACHUSETTS MEMORIAL HOSPITALS

Staff—9. Operations.

NEW ENGLAND BAPTIST HOSPITAL

WALTER B. HOOVER—9. Operations.
F. D. LATHROP—1:30. Operations.

NEW ENGLAND DEACONESS HOSPITAL

WALTER B. HOOVER—9. Operations.
F. D. LATHROP—1:30. Operations.

NEW ENGLAND HOSPITAL FOR WOMEN AND CHILDREN

ELIZABETH DEBLOIS, ISABELLE D. KERR, MARGARET NOYES KLEINERT, and MARION C. SABIA—9. Operations.
ESTHER E. BARTLETT—10. Operations under endotracheal anesthesia.

NEW ENGLAND MEDICAL CENTER
(Boston Floating Hospital)

W. A. MCCOLL—2. Adenoid and tonsil problems.

OPHTHALMOLOGY

Monday

MASSACHUSETTS EYE AND EAR INFIRMARY

F. H. VERHOEFF and WILLIAM P. BEETHAM—2:15. Dry Clinic: Cataract surgery.

Scientific Exhibit—Daily

T. L. TERRY. Pathological laboratory open for inspection.
DAVID G. COGAN. Howe laboratory and library open.

Tuesday

BOSTON CITY HOSPITAL

JAMES J. REGAN and staff—9. Abnormal fundi.

UNITED STATES NAVAL HOSPITAL

JAMES J. REGAN—9. Demonstrations of technique used in flight physical examinations, with discussions of United States Naval requirements.
VIRGIL G. CASTEN—10:30. Operations.

MASSACHUSETTS EYE AND EAR INFIRMARY

PAUL A. CHANDLER, FRED S. THORNE, TRYGVE GUNDERSEN, and PAUL G. HAIRE—9. Operations.
WILLIAM P. BEETHAM—9. Demonstration: Slit lamp.
PAUL A. CHANDLER—2:15. Dry clinic: Glaucoma, indications for operation.
SAMUEL T. CLARKE—3:15. Dry clinic: Goniotomy; medical treatment of glaucoma.

Wednesday

MASSACHUSETTS EYE AND EAR INFIRMARY

WILLIAM P. BEETHAM, SAMUEL H. WILKINS, BRENDAN D. LEAHEY, and DAVID G. COGAN—9. Operations.
GARRETT L. SULLIVAN—9. Demonstration: Perimetry.
E. B. DUNPHY—2:15. Strabismus, orthoptic training.
VIRGIL G. CASTEN—3:15. Dry clinic: Strabismus, operative treatment.

MASSACHUSETTS MEMORIAL HOSPITALS

WILLIAM D. ROWLAND—9:30. Dry clinic.

Thursday

BOSTON CITY HOSPITAL

JAMES J. REGAN and staff—9. Operations.

CARNEY HOSPITAL

GEORGE GAGLIARDI—2. Dry clinic: Suture and cataract extraction.
LEROY FORD—2. Dry clinic: Orbital abscess in children.
SAMUEL T. CLARKE—2. Contact lenses.

MASSACHUSETTS EYE AND EAR INFIRMARY

EDWIN B. GOODALL, RALPH H. RUGGLES, MERRILL J. KING, and HERMAN GROSSMAN—9. Operations.
VIRGIL G. CASTEN—9. Demonstration: Neuro-ophthalmology.
MERRILL J. KING—2:15. Dry clinic: Retinal separation.
BRENDAN D. LEAHEY—3:15. Dry clinic: Keratoplasty.

MASSACHUSETTS MEMORIAL HOSPITALS

WILLIAM D. ROWLAND, JOSEPH J. SKIRBALL, EDWARD L. PERRY, and RALPH H. HOPKINS—2. Operative clinic: Cataract, glaucoma, and muscles.

ST. ELIZABETH'S HOSPITAL

HUGH DONAHUE—10. Operations: Cataract extraction, decompression operation for primary glaucoma.

Friday

CARNEY HOSPITAL

SAMUEL T. CLARKE and staff—9. Operative clinic.

MASSACHUSETTS EYE AND EAR INFIRMARY

BENJAMIN SACHS and JOHN G. JENNINGS—9. Operations.
Staff—9. Demonstrations.
SAMUEL T. CLARKE. Gonioscopy.
T. L. TERRY—2:15. Dry clinic and lantern demonstrations of pathological specimens.
V. H. KAZANJIAN—3:15. Dry clinic: Ophthalmoplastic surgery.

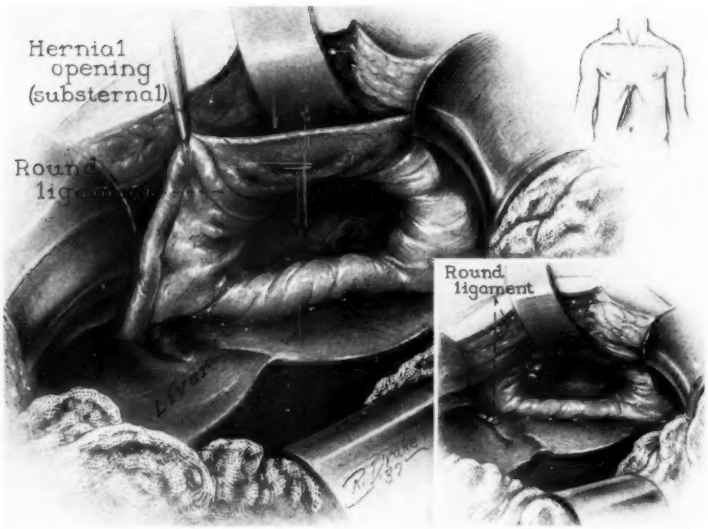

Fig. 20. Case 4. Large subcostosternal opening after removal of herniated colon. Sac of peritoneum containing round ligament extending into thoracic cavity.

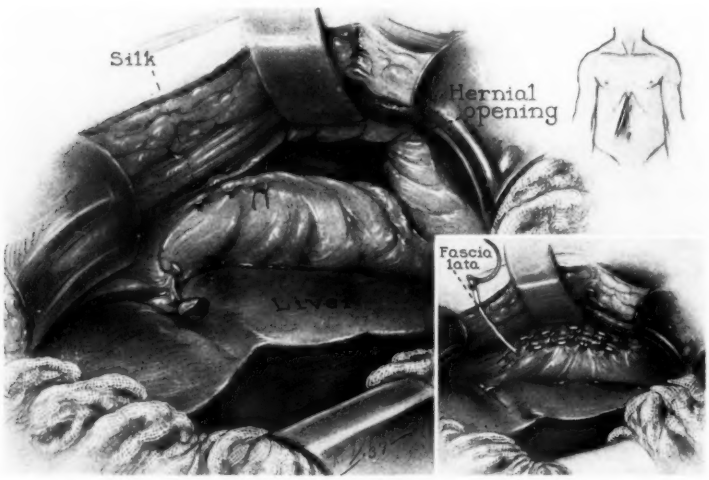

Fig. 21. Case 4. Overlapping closure of hernial orifice with interrupted silk and continuous fascia lata sutures after removal of colon and cutting round ligament.

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